may be important for function (1). In the present study nectadrin glycoforms were purified from thymocytes, splenic lymphocytes, LPS activated B-lymphocytes, erythrocytes and two lymphoma cell lines.The structure of the carbohydrate side chains were analyzed by using a panel of lectins and anti carbohydrate antibodies. A differential reactivity was demonstrated for the lectins SNA, AAA, PNA and SBA and for the 336 mab specific for the HNK-1/L2 carbohydrate structure. The L2 epitope is expressed on B lymphocytes and a large portion of bone marrow neutrophils in the mouse. In search for a potential ligand for nectadrin, we found that mouse P-selectin Ig construct but not control construct was able to bind to some nectadrin glycoforms. The binding correlated with L2 expression.

(1) Kadmon, G., Ekert, M., Sammar, M.; Schachner, M. and Altevogt P. *J. Cell Biol.*, **118**: 1245 – 1258 (1992).

**S8.7**

## Lectin-Glycoconjugate Recognition in Lymphocyte-Endothelial Cell Interactions

*C. Kieda*, V. Denis and M. Monsigny
*Laboratoire des Glycoconjugués et Lectines Endògenes, Centre de Biophysique Moléculaire, CNRS, 1, rue Haute, 45071 Orléans, France.*

Lymphocytes bind to endothelial cells in post capillary veinules of specialized lymph nodes. They do it in a specific way which drives a proper immune response. Among the adhesion molecules involved (1), endogenous lectins along with their specific glycosylated ligands are present on both lymphocytes and endothelial cells. Some lectins, which are dubbed *selectins*, mediate leukocyte binding and rolling.

We used *neoglycoproteins* as tools to show the presence of various membrane lectins among lymphocyte populations (2) and their involvement during *in vivo* homing, on the one hand. On the other hand, a fucose-specific lectin was characterized on the surface of endothelial cells of high endothelial veinules from peripheral lymph nodes. A cell line derived from those cells (SVHEC A10) was established upon transfection with SV40 T antigen gene; this allowed the purification of an Mr 36 000 fucose-binding protein. Expression of this lectin is modulated by various factors unrelatedly to E-selectin. This lectin was not found on the surface of Peyer's patches endothelial cells.

Glycoconjugates containing fucose, present at the surface of an EL4 thymoma derived line EL4-IL2, but not on the original EL4 line, are recognized by the cell surface, as well as, the isolated fucose-specific lectin. Furthermore, glycopeptides from EL4-IL2 cells were purified by affinity chromatography on plant fucose-binding protein, were shown to bind the endothelial cell fucose specific lectin and to inhibit, to some extent, the adhesion of EL4-IL2 cells to SVHEC A10 cells. Pure oligosides (kindly given by G. Strecker, F-Lille) *Fucα*2 Gal were more efficient than Fuc*α*3 GlcNAc and Fuc*α*4 GlcNAC.

(1) Springer, T. A. 1990, *Nature*, **346**, 425 – 434; (2) Kieda *et al.*, 1979, *FEBS Lett.*, **99**, 329 – 332.

**S8.8**

## E-Selectin Dependent *In Vitro* Adhesion of Blood Dendritic Cells to Human Umbilical Cord Endothelial Cells

U. Srinivas[1], M. Larsson[2], A. Lundblad[1] and U. Forsum[2]
*Departments of Clinical Chemistry[1] and Clinical Microbiology[2], Faculty of Health Sciences, 581 85 Linköping, Sweden.*

Peripheral blood dendritic cells (BDC) are potent antigen-presenting lymphoid cells. In the present study, we have examined the *in vitro* adhesion of BDC to human umbilical cord venous endothelial cells (HUVECs) and studied the expression of CD molecules and oligosaccharide haptens on BDC and endothelial cells. Immunohistochemistry showed that BDC were strongly positive for antibodies against HLA DR, CD 11c, CD 18, CD 44 and CD 54 and moderately positive for anti-CD 11 a, CD 31, CD 43 and CD 58. In addition, BDC were moderately positive for anti-Sialyl Lewis$^a$ and strongly positive for anti-Sialyl Lewis$^x$ and CD 77 (Gal*α*1-4Gal*β*1-4Glc). Non-stimulated HUVEC's were positive for anti-CD 29, CD 31 and CD 77. An *in vitro* adhesion assay showed that only a small percentage of radiolabelled BDC bound to non-stimulated HUVECs (16.9 + 1.6%). Stimulation of the HUVEC with Interleukin-1 for 4 h produced an increase in the percentage of adherent radiolabelled BDC (42.3 + 2%). Pre-incubation of HUVEC with antibodies against E-selectin (10 *μ*g/ml) inhibited the binding of radiolabelled BDC to HUVEC whereas pre-incubation of BDC cells with antibodies against CD 54, CD 18, CD 11 b, CD 11 c and Sialyl Le$^x$ did not produce any detectable inhibition. Thus, BDC bind to IL-1 stimulated HUVEC through an E-selectin dependent mechanism. The ligand involved on BDC remains to be clarified.

**S8.9**

## Cancer-Associated Oligosaccharides Stain Selectively Human Cancer Cells

*N. V. Bovin*, T. V. Zemlyanukhina, A. B. Tuzikov, Yu. E. Tzvetkov[1], A. S. Shashkov[1], N. E. Nifant'ev[1], I. V. Abramenko[2] and D. F. Gluzman[2]
*Shemyakin Institute of Bioorganic Chemistry, Moscow, Russia;*
*[1]Zelynsky Institute of Organic Chemistry, Moscow, Russia;*
*[2]Kavetsky Institute for Oncology Problems, Kiev, Ukraine.*

Increased expression of cancer-associated carbohydrate antigens (CAA) is a characteristic feature of cancer cell membrane. Does it mean that CAA interact with the molecules decoding their structural information – endogenous lectins of the same and other cells? Does the level of cancer cell lectins accessible for the interaction decrease or increase and which is their fine carbohydrate specificity?

We've synthesized about 30 probes designed as biotinylated polyacrylamide to which saccharides are attached [1], including GalNAc*α* ($T_n$), GalNAc*α*1-3GalNAc*β* ($F_s$), Gal*β*1-3GalNAc*α* (TF), Gal*β*1-3GalNAc*β* ($T_{\beta\beta}$), Le$^y$, SiaLe$^a$ and Neu5Ac. Most non-reactive were probes Glc*α* and $P_1$ which didn't bind to neither lymphoid nor to myeloid or cancer cells. Human lung and breast cancer cells were stained by the probes more strong than normal cell with preferential staining on plasmatic membrane; most specific towards cancer cells were $T_n$, $F_s$ and SiaLe$^a$ probes; probe Neu5Ac bound selectively to some intercellular organellas and besides more strongly with cancer cells than with normal ones. Simultaneous increase of Gal*β*1-3Gal and its receptor expression on cultured tumour cells and carcinomas was shown in [2], it follows from the present data that expression of CAA-binding molecules has a general character.