respectively. The branch positions were identified with endo-β-galactosidase. The following glycans carrying two GlcNAcβ1-6-branches were also formed in our experiments: Galα1-3'LactNAcβ1-3(GlcNAcβ1-6)'LactNAcβ1-3(GlcNAcβ1-6)'LactNAc and LactNAcβ1-3(GlcNAcβ1-6)'LactNAcβ1-3(GlcNAcβ1-6)'LactNAc.

S1.20

Control of Membrane Glycoprotein Fucosylation

L. I. Stoykova, R. L. Giuntoli, N. Rubinstein and M. C. Glick
Department of Pediatrics, University of Pennsylvania Medical School and The Children's Hospital of Philadelphia, Philadelphia, PA 19104, USA.

Human erythroleukemic (HEL) cells contain high activity for GDP-L-Fuc-*N*-acetyl-β-D-glucosaminide α1→3fucosyltransferase, although Fucα1→3GlcNAc residues are not found on the glycoproteins of HEL cells. To investigate these disparate results it was reasoned that differentiation of HEL cells may bring about glycosylation changes in the membrane glycoproteins. Treatment with phorbol 12-myristate 13-acetate (PMA) differentiates the HEL cells including the ability to adhere within a few hours whereas they normally grow in suspension culture. HEL cells were treated with 0.1 μM PMA, labeled with L-[^{3}H]fucose for two days as adherent cells and harvested. In contrast to HEL cells non PMA-treated, the glycopeptides derived from the PMA-treated cells contained a small amount of Fucα1→3GlcNAc residues as detected with almond α1→3(4)fucosidase. At the same time, α1→3fucosyltransferase activity in the cell extracts was similar with or without treatment. Therefore it is not the activity of α1→3fucosyltransferase *per se* which controls the cell surface expression of Fucα1 3GlcNAc. A study of the requirements of α1→3fucosyltransferase to fucosylate glycoproteins in HEL cells may provide information regarding the activation of ligands for Selectins as well as relate to the ability of other types of cells to form solid tumors at distal sites.

Supported by NIH RO1 CA 37853 and Travel Award from Society for Complex Carbohydrates (L.I.S.)

S1.21

Characterization of a Rat Corpus Sulfotransferase for the 6-*O*-Sulfation of β-D-*N*-Acetylglucosamine Residues on Oligosaccharides

Y. Goso and K. Hotta
Department of Biochemistry, Kitasato University School of Medicine, Sagamihara, 228 Japan.

The oligosaccharides of mucin in gastrointestinal tract are often sulfated. The functions of sulfate residues on the oligosaccharides have yet to be determined in detail but their strong acidic properties may importantly affect mucin molecules. In the rat corpus , at least two types of sulfation on mucin oligosaccharides have been demonstrated (Goso & Hotta (1989) *Biochem. J.* **264**, 805 – 812.). Their occurrence was suggested by the finding that oligosaccharides contained Galβ1-4GlcNAc(6-SO_4) or terminal GlcNAc(6-SO_4) sequences. To determine the molecular basis for production of these sulfated sequences, we have characterized sulfotransferases in the rat corpus microsome. Analysis using simple saccharide acceptors (GlcNAcβ-*O*-Me, GlcNAcα-*O*-Me, GlcNAcβ1-3Galβ-*O*-Me, Galβ1-4GlcNAcβ-*O*-Et, Galβ1-4GlcNAcβ1-6Gal, etc.) indicated an enzyme to transfer sulfate residues toward terminal but not internal β-D-GlcNAc residues of saccharides. Sulfate was not transferred toward α-D-GlcNAc residues even when located at the terminal position. Analysis of the products from saccharide acceptors bearing terminal GlcNAc residue showed the 6-*O*-position of GlcNAc residue to be sulfated by enzyme reaction. 6-*O*-Sulfated GlcNAc residues are present on mucin oligosaccharides and thus this enzyme would appear to transfer sulfate residues toward mucin oligosaccharides. The small degree of sulfate transfer toward internal GlcNAc residues in oligosaccharides by this enzyme may be indication that the Galβ1-4GlcNAc(6-SO_4) sequence is produced by the transfer of the Gal residue to the GlcNAc(6-SO_4) sequence. Other enzyme properties are as follows: (i) optimum pH of this enzyme is about 7.5, (ii) the enzyme does not require divalent cations and (iii) Triton X-100 stimulates enzyme activity.

S1.22

Sialyltransferase Activities in FR3T3 Cells Transformed with *ras* Oncogene : Decreased CMP-Neu5Ac : Galβ1-3GalNAc α-2,3-Sialyltransferase

P. Delannoy, H. Pelczar, V. Vandamme and A. Verbert
Laboratoire de Chimie Biologique, UMR du CNRS n°111, Université des Sciences et Technologies de Lille, 59655 Villeneuve d'Ascq, France.

We have investigated the activity of CMP-Neu5Ac:Galβ1-3GalNAc α-2,3-sialyltransferase (EC 2.4.99.4) in FR3T3 cells transformed by the Ha-*ras* oncogene in which we had previously demonstrated the higher expression of the β-galactoside α-2,6-sialyltransferase (EC 2.4.99.1) (Le Marer *et al.*, 1992, *Glycobiology* **2**, 49 – 56). We clearly demonstrate using different acceptors that the presence of the activated *ras* gene decreased 4 fold the activity of α-2,3-sialyltransferase specific for *O*-glycan core 1. Based on the kinetic parameters and on mixing experiments, we can assume that this decreased enzymic activity reflects a decrease of the number of active *O*-glycan α-2,3-sialyltransferase polypeptide in *ras*-transformed cells rather than the occurrence of an inhibitor in these cells. However, no change in the binding of Peanut agglutinin was observed on the cell surface of *ras*-transformed FR3T3 suggesting that no change in the sialylation of *O*-glycan core 1 appeared in these cells. This suggests that, while the activity of the α-2,3-sialyltransferase was 4 fold decreased in *in vitro* assays, the remaining activity can be sufficient to maintain the same level of sialylation of endogenous acceptor.

S1.23

Investigation of the Sialyltransferases Present in Two Populations of Ehrlich Ascites Tumor Cells

S. Shigeta[1] and I. J. Goldstein
Department of Biological Chemistry, University of Michigan, Ann Arbor, MI, 48109, USA.
[1] *Hiroshima University, Hiroshima, Japan.*

The sialyltransferase activities responsible for the synthesis of *O*-linked and *N*-linked sialoglycoproteins of two populations of Ehrlich ascites tumor cells (EAT-wt and EAT-c cells) were