Enzymatic Transfer of a Preassembled Trisaccharide Antigen to Cell Surfaces Using a Fucosyltransferase*

Geeta Srivastava, Kanwal J. Kaur, Ole Hindsgaul†, and Monica M. Palcić‡

From the Department of Chemistry, University of Alberta, Edmonton, Alberta, T6G 2G2 Canada

(Received for publication, May 14, 1992)

The Lewis α1→3/4-fucosyltransferase (Le-FucT) is known to fucosylate both Type I (βGal(1→3)/βGlcNAc) and Type II (βGal(1→4)/βGlcNAc) sequences even when these are sialylated at OH-3 or fucosylated at OH-2 of the terminal Gal residues. These acceptor sequences are ubiquitous on mammalian cell-surface glycoproteins and glycolipids. The Le-FucT enzyme is therefore a potential candidate as a universal reagent for the modification of cell surfaces.

We have found that a readily accessible, partially purified Le-FucT from human milk, which normally uses GDP-fucose (a 6-deoxy sugar) as the donor for the transfer of a single fucose residue, will also transfer a fucose residue substituted on C-6 by a very large sterically demanding structure, in this instance, a synthetic blood group antigen. As a demonstration of the ability of the Le-FucT to modify glycoconjugates in a mild and specific manner, we chemically synthesized the complex sugar-nucleotide αGal(1→3)[αFuc(1→2)]βGal-O-(CH₂)₆COH-NH₂-L-fucose-GDP (13) which is a GDP-fucose analog where the human blood group B trisaccharide antigen is covalently linked to C-6 of fucose through an amino group. It is shown that, in enzyme-linked immunosorbent assays, the Le-FucT uses both immobilized βGal(1→3)/βGlcNAc-bovine serum albumin conjugates and fetuin as acceptor substrates and renders them blood group B active as detected by a monoclonal anti-B blood-grouping antibody. The fucose residue to which the B-trisaccharide is linked therefore becomes covalently attached to the acceptor oligosaccharide chains of those glycoproteins. Incubation of type "O" erythrocytes with the Le-FucT and complex donor 13 results in the covalent transfer of αGal(1→3)[αFuc(1→2)]βGal-O-(CH₂)₆COH-NH₂-L-Fuc to cell-surface acceptors since the cells become phenotypically "B" and are agglutinated by the same antibody. It is proposed that the Le-FucT represents a powerful new tool with the ability to label animal cell surfaces with preassembled oligosaccharide and possibly also other complex recognition markers.

Elucidation of the biological roles of specific cell-surface glycosylation remains a subject of intense investigation. Specific changes in the detailed structures of cell-surface carbohydrates have been extensively described in, among other cases, cell differentiation, development, and tumor progression (1–3). While changes in their structure can be documented with good accuracy, it remains much more difficult to establish a biological function or significance (if any) to apparently regulated appearances of specific cell-surface carbohydrate sequences. The potential importance of specific glycosylation is highlighted by recent reports describing the essential role of sialylated and fucosylated tetrasaccharide antigens (sialyl-Leα and sialyl-Leβ) in the adhesion of neutrophils to endothelial cells, an example of carbohydrate-mediated cell-cell recognition which, in this instance, appears to be the first step in initiating an acute inflammatory response (4–6).

Well characterized oligosaccharides, most reliably available through chemical synthesis, are important as tools in obtaining evidence for the function of a specific cell-surface carbohydrate sequence. The most common applications for such compounds are as inhibitors of the binding of proteins or cells with the natural glycoconjugate ligand. If inhibition is observed, then that particular carbohydrate sequence becomes a candidate as a physiologically relevant recognition marker, and further experiments can be designed to assess whether the recognition is in fact functional (7). In the work reported here, we decided to investigate whether similar synthetic oligosaccharides could not also be used in an alternate experiment where they could be added in a mild and specific manner to intact glycoconjugates or living cells. If this became feasible, then postulated complex carbohydrate recognition markers could be added in a controlled fashion to proteins or cells which are completely devoid of related structures. The resulting panel of “sugar-tagged” proteins or cells should be unique and invaluable tools for dissecting the biological function of specific oligosaccharide sequences.

In principle, there are two major chemical approaches available for tagging soluble as well as cell-surface glycoproteins with synthetic or isolated oligosaccharides. In the first type of approach, reactive groups on the protein (usually side chain amino, carboxyl, or sulfhydryl) are covalently attached to oligosaccharides by reductive amination or using chemically activated derivatives of the oligosaccharides. This results in alkylation or acylation of the protein. Such methods can be useful but clearly suffer from the disadvantage that peptide chains are actually chemically altered. There can be multiple random substitutions if there are multiple reactive groups on the proteins, and all cell-surface proteins can react. The second approach involves oxidation of the sugar chains of cell-surface glycoproteins (or glycolipids) followed by reductive amination of an amino derivative of the oligosaccharide to be attached. The oxidation is usually effected with periodate or, in a milder procedure, treatment of cells with sulfidase followed by galactose oxidase and then the reductive amination (8). Such methods, when applied with care, cause minimal alteration of peptides, but the chemistry remains vig-
orous (both oxidizing and reducing) by biological standards. Also, existing carbohydrate sequences are either modified or destroyed.

We chose to develop an alternate enzymatic approach using a glycosyltransferase to transfer potential oligosaccharide recognition markers. Glycosyltransferases have in the past been used effectively to add sugars to both glycoproteins and cell-surface glycoconjugates (9-11). In such experiments, single sugar residues are added, from the appropriate sugar nucleotides, in a stepwise fashion. To build up a complex oligosaccharide, a series of glycosyltransferases is therefore required. A more important limitation of this stepwise approach, however, is that a sequential series of glycosylations cannot likely be driven to completion and, as a result, a series of partial structures will invariably be present on the target glycoproteins or cells that have been constructed. We report here that a partially purified (α1→3/4)-fucosyltransferase (Le-FucT) from human milk (12, 13), which bio-synthetically uses GDP-fucose as the donor to transfer single fucose residues, is capable of transferring large preassembled oligosaccharide antigens when these are covalently attached through C-6 of the fucose residue. Using this enzyme, the sugar chains of glycoconjugates, both free and membrane-bound, can thereby be tagged with completed carbohydrate recognition markers. The decision to investigate whether the human Le-FucT could be used to add large oligosaccharides to cell surfaces was based on three considerations. Firstly, the enzyme can be easily partially purified from human milk (12, 13). It has also been cloned (14) which augers well for more ready availability in the near future. Secondly, fucosyltransferases are the only glycosyltransferases which transfer a sugar having the L absolute configuration. It therefore seemed at least plausible that fucosyltransferases in general might not have highly evolved and specific recognition sites for the sugar being transferred since the remaining mammalian sugars in the Leloir pathway all have the D configuration. The Le-FucT has indeed been shown (15) to transfer both 3-deoxy sugars and arabinoses, a fucose analog where the 6-methyl group is missing. Sialyltransferases are also candidates in this regard since they are the only ulosonic acids transferred in mammalian biosynthesis. In general, sialyltransferases do accept modifications on the sugar nucleotide, CMP-sialic acid, and several modified sialyl residues have been added enzymatically to galactose-terminated glycoconjugates (16). The third and most important consideration, however, was that a large number of the carbohydrate structures commonly found on cell-surface mammalian glycoproteins and glycolipids can act as acceptors for the Lewis enzyme. The structures of these ubiquitous (1-3, 17, 18) acceptor sequences and the products that would form on their reaction with GDP-fucose catalyzed by the Le-FucT are shown in Fig. 1. This figure also summarizes our strategy for tagging cell-surface glycoconjugates. This strategy involves covalent attachment of synthetic oligosaccharides directly to the fucose residue of GDP-Fuc to produce a complex analog of that sugar nucleotide. The hope was that the FucT would still recognize this analog as a donor substrate thereby covalently attaching the oligosaccharide to the cell surface via a fucose “spacer.” This proved to be the case.

**EXPERIMENTAL PROCEDURES**

**Materials**

Fetuin (fetal calf serum, Type IV), alkaline phosphatase conjugate of goat anti-mouse IgM (μ-chain-specific), alkaline phosphatase substrate tablets containing 5 mg of p-nitrophenyl phosphate, Tween 20, and bovine serum albumin were obtained from Sigma. Anti-B monoclonal antibodies, either affinity-purified in 1% BSA1 in PBS (Synaff) or Syntype B were from Chembiomed, Edmonton. Partially purified α(1→3/4)fucosyltransferase was isolated from human milk by precipitation with 65% (NH4)2SO4, and chromatography on CM-Sephadex C-50 (0.2 M NaCl eluent) and assayed as previously described (13). Type I BSA conjugates (αGal(1→3)βGlCNac-O-(CH2)nCONH2)n-α7-BSA) were available from previous work (21). Removable flat-bottomed wells of Immulon 2 were from Dynatech. The following buffers were used: PBS, 7.3 mM NaH2PO4, 2.2 mM KH2PO4, 0.09% NaCl, and 15 mM NaHCO3, pH 7.4; PBST, PBS with 0.05% Tween 20; cell incubation buffer, 25 mM sodium cacodylate, pH 6.8, with 7 mM MnCl2, 75 mM NaCl, 100 mM glucose, and 10 mg/ml bovine serum albumin. All reagents used for chemical synthesis were from either Aldrich or Sigma.

**Chemical Synthesis of GDP-Fuc Derivatives**

The general synthetic scheme is modelled after a previously reported synthesis of GDP-Fuc and two of its analogs (15). Details on the chromatography and spectroscopic characterization of intermediates are discussed in detail in that publication. The structures of synthetic intermediates are shown in Fig. 2. The following solvents used here have been designated by letters: A, benzene/methanol, 9:1 (v/v); B, benzene/methanol, 12:1 (v/v); C, ethyl acetate/hexane, 1:2; D, dichloromethane/methanol/water, 60:35:6; E, 2-propanol/water/

---

1 The abbreviations used are: BSA, bovine serum albumin; PBS, phosphate-buffered saline; ELISA, enzyme-linked immunosorbent assay.
ammonium hydroxide. 7:4:1, F, 2-propanol/water/ammonium hydroxide, 1:10:2. G, 2-propanol/water/ammonium hydroxide, 7:3:1. NMR spectroscopy was performed on a Bruker 360 WM spectrometer operating at ambient temperature.

**Synthesis of GDP-6"-Fuc (13)**

1,2,3,4-Di-O-isopropylidene-α-L-galactopyranose (6)—To anhydrous zinc chloride (2.2 g) in dry acetone (45 ml) was added concentrated sulfuric acid (72 µl). Then powdered anhydrous t-galactose (1.3 g, 10 mmol) was added quickly, and the reaction mixture was stirred for 10 min. A suspension of sodium hydrate carbonate (3.6 g) in water (6.3 ml) was then added in portions. The suspension was filtered with suction, and the precipitate was washed several times by suction in acetone and filtering. The filtrate and washings were combined, and the solution and the acetone were evaporated under diminished pressure. The residue was extracted with ether (3 × 10 ml), dried over anhydrous sodium sulfate, filtered, and evaporated to dryness. The residue was dissolved in uacuo in 2-propanol/water/ammonium hydroxide (100 ml), the solvent was dried and evaporated. The residual syrup was then added, and stirring was continued for 10 days, then concentrated in uacuo to provide 6 (R = 0.25, solvent A) was obtained as a syrup (2.1 g, 80.8%) [δ 6.40 (d, 1 H, J1,2 = 5.0 Hz, H-1), 4.62 (dd, 1 H, J1,2 = 5.0Hz, J1,3 = 8.0 Hz, H-1), 1.43, 1.41, 1.40, 1.39, 1.38, 1.37, 1.36, 1.35, 1.34, 1.33 (each 3 H, CH3)].

The acetylated mixture was diluted with dichloromethane (100 ml), washed with cold 1M HCl for 15 h, then concentrated in uacuo to provide compound 7 (750 mg, 2.63 mmol) was dissolved in trifluoroacetic acid (100 ml), the solvent was evaporated and the residue was purified by chromatography on silica gel (solvent A) to provide compound 7 as the triethylammonium salt (0.054 mmol) and guanosine 5′-monophosphate morpholidate (46.5 mg, 0.064 mmol) in anhydrous pyridine (10 ml) were concentrated in uacuo to provide 7 (R = 0.47, solvent B) was obtained as a syrup (195 mg, 94%) after lyophilization; [1H NMR (D2O) 6 4.79 (dd, 1 H, J1,2 = 8.0 Hz, H-1), 3.87 (d, 1 H, J1,2 = 8.0 Hz, H-4), 3.66 (dd, 1 H, J1,2 = 10.0 Hz, H-3, 3.50 (dd, 2 H, J1,2 = 8.0 Hz, H-3); 13C NMR (D2O) 6 96.3 (C-1), 100.8 (C-2), 106.1 (C-3), 114.0 (C-4), 140.7 (C-5), 164.0 (C-6), 87.4 (C-7), 64.5 (C-8), 85.3 (C-9), 63.7 (C-10), 26.0 (C-11), 26.0, 26.0, 25.9, 24.9, and 24.3 (each CH).] 6-Azido-1,2,3,4-tetra-O-acetyl-6-deoxy-α-L-galactopyranose (8)—Compound 7 (11 g, 4.23 mmol) was added to the residue and re-evaporated. The residue was then added, and stirring was continued for 15 min at room temperature. The acetylated mixture was diluted with diethyl ether (100 ml) and washed with cold 1 M HCl for 15 h, then concentrated in uacuo to provide 8 (R = 0.43, solvent C) as an α,β-mixture (850 mg, 87%). [1H NMR (CDCl3) 6 δ 4.60 (d, J1,2 = 3.2 Hz, H-1), 5.72 (d, J1,2 = 8.0 Hz, H-1), 4.23 (dd, J1,2 = 6.5 Hz, H-5a), 3.98 (dd, J1,2 = 6.5 Hz, H-5b), 2.19, 2.18, 2.17, 2.15, 2.06, 2.05, 2.03, and 2.02 (each 3, COCH3).] 6-Azido-1,2,3,4-tetra-O-acetyl-6-deoxy-α-L-galactopyranose Phosphate Triethylammonium Salt (9)—Tetraphenylborate was obtained as a white powder after converting it into triethyl ammonium salt by passage through Dowex 50-X8 (Et3NH+) ion exchange resin by guest on March 24, 2020 http://www.jbc.org/ Downloaded from
5'-monophosphate morpholidate to yield the GDP-derivative. Details of the synthesis of 2 and other GDP-Fuc analogs not described here will be reported elsewhere. The final 6-O-propyl-GDP-1-l-galactose (2) had the following spectral characteristics: 

\[
\begin{align*}
\text{H NMR} (D_2O) & \delta 8.10 \\
(1, \text{ H, H-8 guanine}) & 5.93 \\
(1, J = 6.0 Hz, H-1 ribose) & 4.88 \\
(\text{dd}, J = 8.0 Hz, H-1, 3'Fuc) & 1.55 \\
(2, H, OCH_2CH_2CH_3) & 0.86 \\
(2, H, J = 7.0 Hz, OCH_2CH_2CH_3).
\end{align*}
\]

Comparison of Transfer of GDP-Fuc with GDP-6-O-propyl Gal

Preparative fucosylation was performed essentially as previously described (21). Briefly, to a mixture of acceptor 3 (1.4 μmol), GDP-Fuc (1, 1.4 μmol), and GDP-6-O-propyl-galactose (2, 1.4 μmol), ADP (0.5 μmol), and NaCN (4 μmol) was added 1.7 milliliters of Le-FucT in 0.5 ml of sodium cacodylate buffer (25 mM, pH 6.5) containing MnCl_2 (5 mM). After 28 h at 37 °C, the solution was diluted with water, and the hydrophobic products were isolated on a C-18 Sep-Pak cartridge (21). The H NMR spectrum of the crude product confirmed that complete fucosylation of disaccharide 3 had occurred, and integration of the signal for the known (21) Le? trisaccharide versus the new signals for the trisaccharide containing an O-propyl group indicated that the ratio of 4 to 5 was approximately 2:1.

The Use of ELISA to Detect the Transfer of Fucose Analogos to Immobilized Glycoconjugates: ELISA Plate Coating with Type 1 BSA Conjugates (βGal(1→3)βGlcnAc-OCH_2CO-NH-BSA)

Microtiter plates were coated by incubating with 100 μl of BSA-glycoconjugate (20 μg/ml) or fetuin (50 μg/ml) in 50 mM potassium phosphate buffer, pH 7.5, containing 5 mM MgCl_2 and 15 mM NaN_3. After 16 h at ambient temperature (4 °C for the fetuin plates), the solution was aspirated and replaced with 100 μl of 5% BSA in PBS. After 4 h, this solution was removed, and the wells were washed three times with 200 μl of PBST and once with 200 μl of H_2O before adding p-nitrophenyl phosphate (1.0 mg/ml in 50 mM potassium phosphate buffer, pH 9.8, containing 0.5 M diethanolamine-HCl buffer, pH 9.8, containing 1% BSA and 0.5 mM MgCl_2). The increase in absorbance at 405 nm with background subtraction of the control reactions lacking either enzyme or donor was assayed. After incubation, the reaction mixtures were removed, and the wells were washed three times with 200 μl of PBST. Anti-B antibody (Synaff) was diluted 1:1000 in 1% BSA/PBST, and 100 μl was added to the microtiter wells. After 2 h at ambient temperature, the antibody solution was aspirated, washed 3 times with 200 μl of PBST, and then incubated with 100 μl of the alkaline phosphatase-conjugated goat anti-mouse antibody (1:1000 dilution in 1% BSA/PBST) for 2 h at ambient temperature. Solutions were aspirated, and the wells were washed 3 times with 200 μl of PBST and once with 300 μl of H_2O before adding p-nitrophenyl phosphate (1.0 mg/ml in 1 M diethanolamine-HCl buffer, pH 9.8, containing 1% BSA and 0.5 mM MgCl_2). The increase in absorbance at 405 nm with background correction at 650 nm was monitored with a Molecular Devices Thermax microplate reader. Data acquisition was controlled by a Macintosh SOFTmax program, and the absorbance readings reported were taken after 120 min of color development.

Transfer of the B-active Trisaccharide to Red Cells Detected by Agglutination: Cell Incubations

Fresh red blood cells type O Le° Leh), were collected by venipuncture and treated with 1/10 volume of 38% sodium citrate as an anticoagulant. 100 μl of whole red cells were washed twice with 1 ml of cell incubation buffer by suspending the cells in buffer in 1.5-mlmicrocentrifuge tubes, centrifuging for 15--30 s, and removing supernatant buffer with a pipette. Packed cells (100 μl) were incubated with 62 micromolars of α(1→3/4)fucoyltransferase and 116 μM donor 13 in 120 μl of cell incubation buffer. Control reaction mixtures contained only enzyme or donor in 120 μl of cell incubation buffer. After 15 h at 37 °C, the red cells were washed twice with PBS, spun in Microfuge tubes for 15 s after each wash, and resuspended in 1 ml of PBS. For hemagglutinations, 100 μl of a 2% cell suspension were mixed with 100 μl of Syntype anti-B antibody serially diluted in PBS, the mixtures were immediately spun at 1000 × g for 15 s, and scored by the method of Marsh (25).

RESULTS

The Le-FucT Can Transfer a Fucose Residue Chemically Substituted at C-6—The first experiments performed were to establish that GDP-Fuc (1) could be substituted by a sterically demanding group without destroying its activity as a donor for the Lewis FucT. For ease of chemical synthesis, only substitution at C-6 was examined and GDP-(6-O-propyl)-L-galactose (2) was synthesized. The Type 1 disaccharide βGal(1→3)βGlcnAc-OR (3) (19) was used as the acceptor since we have previously demonstrated (13, 15) its conversion to the Le° trisaccharide βGal(1→3)(αFuc(1→4))βGlcnAc-OR (4) using an identical preparation of the FucT (Fig. 3). A competition experiment was set up where equal amounts of GDP-Fuc (1) and its 6-O-propyl analog 2 were incubated with acceptor 3 and the FucT. The ratio of the trisaccharide products (4 and 5) was determined to be near 2:1 from the 'H NMR spectra of the crude products. Analog 2 therefore remains very active as a donor for the FucT, despite the introduction of a large O-propyl group on C-6 (Fig. 3), and the preparation of a more complex analog could therefore be justified.

Covariant Attachment of the Blood Group B-active Trisaccharide, αGal(1→3)[αFuc(1→2)]βGal, to C-6 of the Fucose Residue in GDP-fucose Does Not Destroy Its Donor Activity—Since a substitution as large as an O-propyl group could be made on C-6 of the Fuc residue of GDP-Fuc, the conclusion was that this part of the molecule did not interact with the enzyme active site. It thus seemed probable that much larger groups could also be attached to this position. To examine

FIG. 3. Substrates for the Le-FucT. A, comparison of the structures of GDP-Fuc (1) and GDP-1-L-galactose (2). B, the solution reaction catalyzed by the Le-FucT. C, the structure of GDP-"B" (13).
this possibility, we synthesized 6-amino-β-fucose-1-phosphate (10) where the 6-amino group could now easily be derivatized by simple acylation. The 8-methoxycarbonyloctyl derivative of a blood group B active trisaccharide, αGal[1→3](αFuc[1→2])βGal-O-(CH₂)₆COOMe (11), available from previous work (20), was then used to acylate the 6-amino group following established procedures. Pyrophosphate formation to provide the GDP-“B”-Fuc structure 13 (Fig. 3) was effected as previously described (15) for other analogs of GDP-fucose.

GDP-“B”-Fuc (13) was shown to be a donor for the Le-FucT using an ELISA assay system. First, the synthetic conjugate of βGal(1→3)βGlCNAC-O-(CH₂)₆COOMe-NH-BSA (21), previously shown to be an acceptor for the Le-FucT in an ELISA using GDP-Fuc as the donor and a monoclonal anti-Le” as the detecting antibody (21), was coated on microtiter plates. The coated plates did not bind a commercial anti-B bloodtyping antibody, as expected. When these coated plates were incubated with the FucT in the presence of GDP-“B”-Fuc (13), the plates acquired B activity, as detected by the anti-B antibody (22), despite repeated washings (A₄₀₅ = 0.70, 1.33, and 2.63 for 2-, 6-, and 21-h incubations, respectively). Omission of either the FucT or donor 13 led to B-inactive plates (A₄₀₅ = 0.063). The conclusion was therefore reached that the B-trisaccharide was covalently attached to the conjugate on the plate. A detailed investigation of the donor properties of GDP-Fuc (13), previously shown to be an acceptor for the Lewis α(1→3/4)Fucotransferase. Most glycoproteins, and possibly all cell surfaces, possess some of the FucT acceptor sequences shown in Fig. 1, and it should consequently be possible to label these glycoproteins and cells in this very simple and direct manner. Clearly, the new linkage, via a fucose “spacer,” is unnatural, and the labeling strategy reported here should not be considered as a viable method for remodeling the sugar chains of glycoconjugates for in vivo use. We expect that the major use of this labeling strategy will be in rapidly assessing whether specific carbohydrate sequences are sufficient to target glycoproteins or cells to carbohydrate-recognizing receptors and to assist in the discovery of such receptors. The labeling strategy should

\[ \text{Fucose “spacer”} \]

**FIG. 4.** Schematic representation of the conversion of blood group O erythrocytes to type B by incubating the cells with Le-FucT and GDP-“B”-Fuc (13). The postulated positions of attachment of the B-trisaccharide through a fucose spacer to Type I and Type II chains are shown.

**FIG. 5.** Agglutination of blood group O cells by an anti-B antibody after incubation with the Le-FucT and GDP-B (13). Duplicates are shown. In the right-hand pair, the O cells were incubated with Le-FucT, but the donor (13) was omitted. In the center pair, the cells were incubated with the donor (13), but the Le-FucT was omitted. In the pair to the left, the cells were incubated with both the Le-FucT and donor (13). Only the latter pair was agglutinated by the anti-B antibody.
be attractive since, once the GDP-Fuc analog has been synthesized, the procedure involves only incubation under neutral conditions of the FucT, donor, and glycoconjugate to be derivatized. The major impediment to this labeling approach remains the synthesis of the complex sugar nucleotide analogs which we are attempting to simplify. The exciting possibility that peptides, or even large proteins, attached to the fucose residue of GDP-fucose might be transferred is also under investigation.

Acknowledgments—We thank A. Heather Good for supplying the red blood cells and carrying out the hemagglutinations and Lakhu M. Keshvara for performing the ELISA test for B activity.

REFERENCES
1. Hakomori, S.-I. (1989) Adv. Carcin Res. 52, 257-331
2. Kobata, A. (1988) Biochimie 70, 1575-1585
3. Dennis, J. W. (1992) in Cell Surface Carbohydrates and Development (Fukuda, M., ed) pp. 161–194, CRC Press, Boca Raton, FL
4. Phillips, M. L., Nudelman, E., Gaeta, F. C., Perez, M., Singhal, A. K., Hakomori, S.-I., and Paulson, J. C. (1990) Science 250, 1130-1132
5. Walz, G., Aruffo, A., Kolanus, W., Bevilaqua, M., and Seed, B. (1990) Science 250, 1132-1135
6. Brandley, K. R. (1991) Semin. Cell Biol. 2, 281-287
7. Varki, A. (1991) Trends Glycosci. Glycotechnol. 3, 122-129
8. Gamberg, C. G., and Tolvanen, M. (1988) Anal. Biochem. 170, 520-527
9. Beyer, T. A., Sadler, J. E., Roarick, J. L., Paulson, J. C., and Hill, R. L. (1981) Adv. Enzymol. Relat. Areas Med. Biol. 52, 24-173
10. Kelm, S., Shukla, A. K., Paulson, J. C., and Schauer, R. (1986) Carbohydr. Res. 149, 53-64
11. Berch, M. L. E., Hubbard, C. S., and Robbins, P. W. (1988) Burnaby Rep. 29, 59-68
12. Przibis, J. P., Monson, D., Dolman, M., Beyer, T. A., and Hill, R. L. (1981) J. Biol. Chem. 256, 10456-10465
13. Palcic, M. M., Venot, A. P., Ratcliffe, R. M., and Hindsgaul, O. (1989) Carbohydr. Res. 190, 1-11
14. Klukowska, J. F., Larsen, R. D., Nair, R. P., and Lowe, J. B. (1990) Genes & Dev. 4, 11288-12039
15. Gokhale, U. B., Hindsgaul, O., and Palcic, M. M. (1990) Canad. J. Chem. 68, 1063-1071
16. Gross, H. J., Runci, J. C., Paulson, J. C., and Breuer, R. (1987) Eur. J. Biochem. 166, 595-602
17. Montreuil, J. (1982) in Comprehensive Biochemistry (Neuberger, A., and van Deenen, L. L. M., eds) Part III, Vol. 19B, pp. 1-188, Elsevier Science Publishers B. V., Amsterdam
18. Rademacher, T. W., Parekh, R. B., and Dwek, R. A. (1988) Glycobiohgy 87, 781-838
19. Lemieux, R. U., Bundle, D. R., and Baker, D. A. (1975) J. Am. Chem. Soc. 97, 4076-4083
20. Lemieux, R. U., Venot, A. P., Spohr, U., Bird, P., Mandel, G., Morishima, N., Hindsgaul, O., and Bundle, D. R. (1985) Canad. J. Chem. 63, 2664-2668
21. Palcic, M. M., Ratcliffe, R. M., Lamontagne, L. R., Good, A. H., Alton, G., and Hindsgaul, O. (1990) Carbohydr. Res. 196, 133-140
22. Keshvara, L. M., Newton, E. M., Good, A. H., Hindsgaul, O., and Palcic, M. M. (1992) Glycoconj. J. 9, 16-20
23. Townsend, R. R., Hardy, M. R., Wong, T. C., and Lee, Y. C. (1986) Biochemistry 25, 5716-5725
24. Bendiak, B., Harris-Brandts, M., Michnick, S. W., Carver, J. P., and Cumming, D. A. (1989) Biochemistry 28, 6491-6499
25. Walsh, W. L. (1972) Transfusion 12, 552-351
26. Pinto, M. R., and Bundle, D. R. (1985) Carbohydr. Res. 124, 313-318
27. Palcic, M. M., Heerze, L. D., Pierce, M., and Hindsgaul, O. (1990) Glycoconj. J. 5, 49-63
Enzymatic transfer of a preassembled trisaccharide antigen to cell surfaces using a fucosyltransferase.

G Srivastava, K J Kaur, O Hindsgaul and M M Palcic

J. Biol. Chem. 1992, 267:22356-22361.

Access the most updated version of this article at http://www.jbc.org/content/267/31/22356

Alerts:
- When this article is cited
- When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 0 references, 0 of which can be accessed free at http://www.jbc.org/content/267/31/22356.full.html#ref-list-1