Supplemental Material can be found at:
http://www.jbc.org/content/suppl/2009/05/06/M109.012807.DC1.html

⌘ Author's Choice

THE JOURNAL OF BIOLOGICAL CHEMISTRY VOL. 284, NO. 27, pp. 18537–18544, July 3, 2009
© 2009 by The American Society for Biochemistry and Molecular Biology, Inc. Printed in the U.S.A.

Prolectin, a Glycan-binding Receptor on Dividing B Cells in Germinal Centers*ⓢ

Received for publication, March 6, 2009, and in revised form, April 24, 2009 Published, JBC Papers in Press, May 6, 2009, DOI 10.1074/jbc.M109.012807

Sarah A. Graham[1], Sabine A. F. Jégouzo[1], Sheng Yan[1], Alex S. Powlesland, Jacob P. Brady, Maureen E. Taylor, and Kurt Drickamer[2]

From the Division of Molecular Biosciences, Department of Life Sciences, Imperial College, London SW7 2AZ, United Kingdom

Prolectin, a previously undescribed glycan-binding receptor, has been identified by re-screening of the human genome for genes encoding proteins containing potential C-type carbohydrate-recognition domains. Glycan array analysis revealed that the carbohydrate-recognition domain in the extracellular domain of the receptor binds glycans with terminal α-linked mannose or fucose residues. Prolectin expressed in fibroblasts is found at the cell surface, but unlike many glycan-binding receptors it does not mediate endocytosis of a neoglycoprotein ligand. However, compared with other known glycan-binding receptors, the receptor contains an unusually large intracellular domain that consists of multiple sequence motifs, including phosphorylated tyrosine residues, that allow it to interact with signaling molecules such as Grb2. Immunohistochemistry has been used to demonstrate that prolectin is expressed on a specialized population of proliferating B cells in germinal centers. Thus, this novel receptor has the potential to function in carbohydrate-mediated communication between cells in the germinal center.

Membrane-bound mammalian glycan-binding receptors, often referred to as lectins, are believed to play multiple distinct roles in the immune system, decoding information in complex oligosaccharide structures on cell surfaces and soluble glycoproteins (1, 2). A host of glycan-binding receptors on dendritic cells and macrophages function in pathogen recognition, often resulting in uptake of microbes through endocytic mechanisms. Examples include the mannose receptor, DC-SIGN,[3] langerin, and the macrophage galactose receptor. Glycan-binding receptors can also recognize glycans found on the surfaces of mammalian cells. Some of these receptors, such as the selectins, mediate adhesion between leukocytes and endothelia (3, 4). A small number of receptors, notably members of the siglec family, bind mammalian-type glycans and have been shown to have potential signaling functions (5). While multiple glycan-binding receptors have been described on cells of the myeloid lineage, the complement of such receptors on lymphocytes is much more restricted. The best characterized examples are the T-cell adhesion molecule L-selectin (4) and the B-cell receptor CD22, also designated siglec-2 (5).

Genomic screening for potential glycan-binding receptors has usually been undertaken by initially searching for the presence of one of the several types of structural domains that are known to support sugar-binding activity (6). Knowledge of the structures of multiple families of modular carbohydrate-recognition domains (CRDs) has facilitated identification of proteins with potential sugar-binding activity and can lead to predictions of what types of ligands might be bound. Although the human genome has been extensively screened with profile-recognition algorithms that identify common sequence motifs associated with CRDs, refinements to the genome sequence and improvements in gene-recognition algorithms occasionally result in detection of novel proteins that contain putative CRDs.

We describe a previously undetected glycan-binding receptor identified by re-screening of the human genome and provide characterization of its molecular and cellular properties. Based on its expression in a specialized population of proliferating B cells in germinal centers, we propose that it be designated prolectin. Our results suggest that prolectin functions in carbohydrate-mediated communication between cells in the germinal center.

EXPERIMENTAL PROCEDURES

Prolectin Cloning, Expression, and Purification—The full-length cDNA was amplified from a spleen cDNA library (Clontech) using 40 cycles of PCR with Advantage 2 polymerase mix from Takara and forward primer CCCTGGCTGCCACTTGTCAGGTTC and reverse primer GGGCTTCAACAGGAACATTTCCGC (Invitrogen). The amplified cDNA was isolated by gel electrophoresis and cloned into vector pCRII-TOPO (Invitrogen).

The portion of the cDNA encoding the extracellular domain of prolectin was inserted into the expression vector T5T and expressed in *Escherichia coli* strain BL21(DE3) following the procedure used for DC-SIGN (7). Inclusion bodies isolated by sonication were dissolved in guanidine hydrochloride in the presence of a small amount of 2-mercaptoethanol and renatured by dilution into loading buffer (0.5 M NaCl, 25 mM Tris-Cl, pH 7.8, 25 mM $CaCl_2$) followed by extensive dialysis against the same buffer. Protein from 6 liters of bacterial culture, in a final volume of 500 ml of loading buffer, was isolated on a 10-ml column of mannose-Sepharose (8), which was washed with

* This work was supported by Grant 075565 from the Wellcome Trust (to M. E. T. and K. D.). This work was also supported, in part, by National Institutes of Health Grant GM062116 to the Consortium for Functional Glycomics.
⌘ *Author's Choice*—Final version full access.
ⓢ The on-line version of this article (available at http://www.jbc.org) contains supplemental Table S1.
[1] These authors contributed equally to this work.
[2] To whom correspondence should be addressed: Division of Molecular Biosciences, Dept. of Life Sciences, Biochemistry Bldg., Imperial College, London SW7 2AZ, United Kingdom. Tel.: 44-20-7594-5282; Fax: 44-20-7594-3057; E-mail: k.drickamer@imperial.ac.uk.
[3] The abbreviations used are: DC-SIGN, dendritic cell-specific intercellular adhesion molecule 3-grabbing non-integrin; CRD, carbohydrate-recognition domain; ^{125}I-Man-BSA, iodinated, mannose-conjugated bovine serum albumin.

Downloaded from www.jbc.org by guest, on May 3, 2011

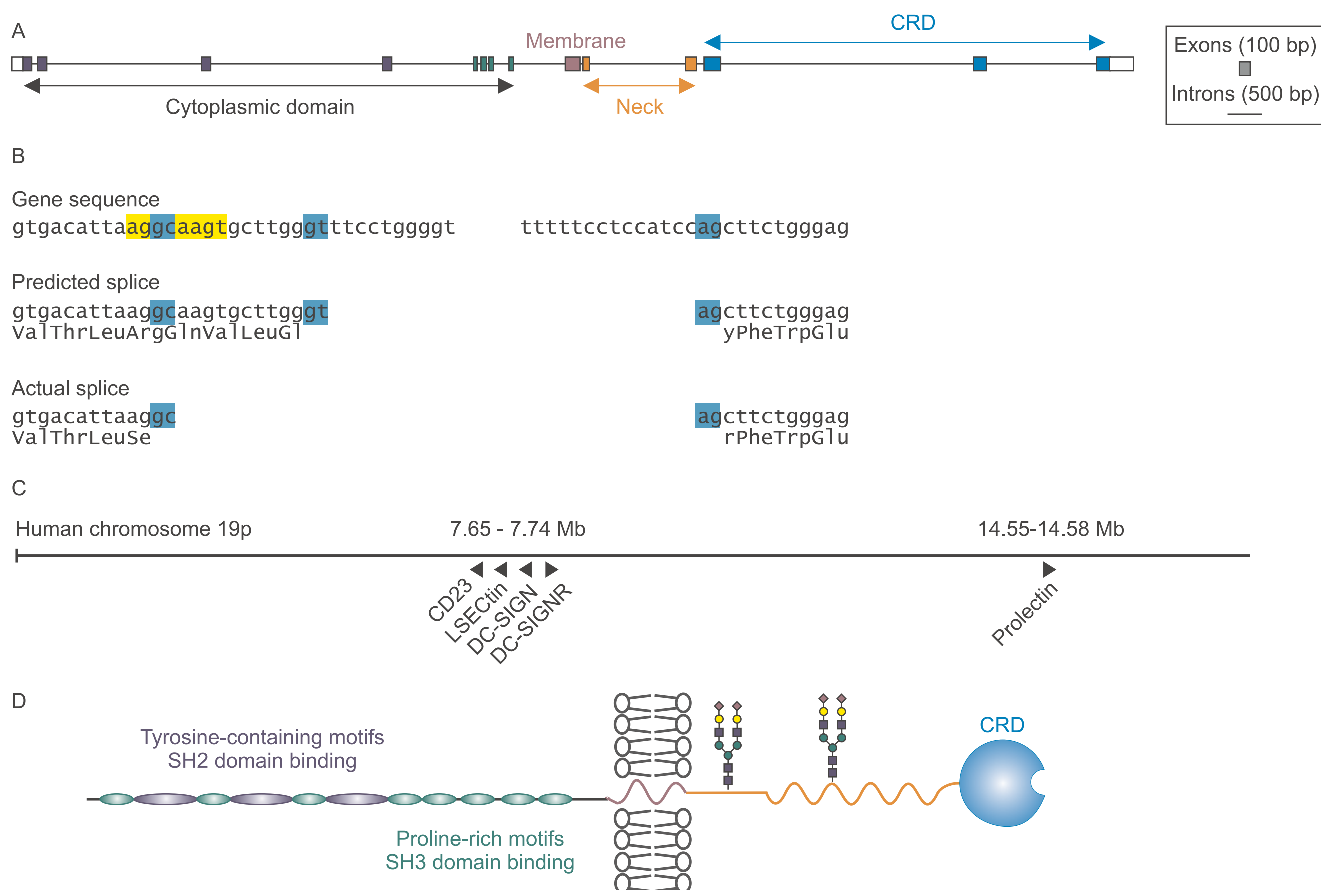


FIGURE 1. **Prolectin gene structure and protein organization.** *A*, scale diagram of the prolectin gene. For clarity, exons are shown expanded 3-fold relative to introns. *B*, details of unusual GC splice site at the 5′ end of the final intron. The predicted GT splice site in the Celera annotation of the genome, GenBank™ accession EAW84437, leads to an insertion of 12 bases corresponding to insertion of 4 amino acids in the predicted protein sequence, but it leaves the reading frame intact so that the conserved residues of the CRD are present. *C*, scale diagram showing the relative positions of glycan-binding receptors containing C-type CRDs encoded on human chromosome 19. *D*, distribution of functional domains in prolectin. Predicted *N*-glycosylation sites are indicated with hypothetical glycans in *stick figures*.

loading buffer and eluted with 2-ml fractions of eluting buffer (0.5 M NaCl, 25 mM Tris-Cl, pH 7.8, 2.5 mM EDTA). Aliquots (25 μl) of fractions were examined by SDS-PAGE (9).

Sugar Binding and Glycan Array Analysis—For glycan array analysis, modified primers were used to append a biotinylation tag Gly-Leu-Asn-Asp-Ile-Phe-Glu-Ala-Gln-Lys-Ile-Glu-Trp-His-Glu after the C-terminal cysteine residue of the CRD. The modified cDNA was inserted into vector T5T, co-expressed with plasmid birA, which encodes biotin ligase (Avidity), and induced in the presence of biotin (10). The monomeric, biotinylated protein, purified on a 10-ml column of mannose-Sepharose as described above for the extracellular domain, was complexed with Alexa-488-labeled streptavidin (Invitrogen) by incubation overnight at a ratio of ~2 mol of CRD to 1 mol of streptavidin subunit. The complex was isolated on a 1-ml column of mannose-Sepharose, which was washed with loading buffer and eluted with 0.5-ml aliquots of elution buffer. The protein was tested against version 3.1 of the glycan array of the Consortium for Functional Glycomics using the standard protocol. The extracellular domain of prolectin was used to coat polystyrene wells, which were used for solid-phase binding competition assays with ^{125}I-Man-BSA (E-Y Laboratories) as the reporter ligand (7). Oligosaccharide ligands were obtained from Carbosynth Ltd.

Analysis in Transfected Fibroblasts—The full-length cDNA was cloned into vector pVcos and used to transfect Ψ-cre packaging cells (11). The resulting pseudoviruses were harvested and used to infect Rat-6 fibroblasts, which were subjected to selection in 400 μg/ml G418. Analysis of endocytic activity with ^{125}I-Man-BSA was conducted as previously described (11). Receptor was isolated from cells by solubilization in lysis buffer (150 mM NaCl, 25 mM Tris-Cl, pH 7.8, 2 mM $CaCl_2$) containing 1% Triton X-100 and protease and phosphatase inhibitors (Set 1 and Set 2 from Calbiochem). After brief sonication and incubation for 30 min on ice, debris was removed by centrifugation for 15 min at 100,000 × *g*, and the supernatant was applied to a 1-ml column of mannose-Sepharose, which was washed with lysis buffer containing 0.1% Triton X-100 and eluted in 0.5-ml fractions with elution buffer containing 0.1% Triton X-100. Fractions were precipitated by addition of trichloroacetic acid to 10% for 10 min on ice, and precipitates were centrifuged for 5 min at 18,000 × *g*, washed twice with 0.5 ml of ethanol:ether (1:1), and dissolved in sample buffer for gel electrophoresis.

Rabbit polyclonal antibodies to prolectin were generated using 1 mg of the bacterially expressed extracellular domain as antigen following the speedy 28-day rabbit polyclonal antibody program from Eurogentec. For affinity purification, an affinity

Downloaded from www.jbc.org by guest, on May 3, 2011

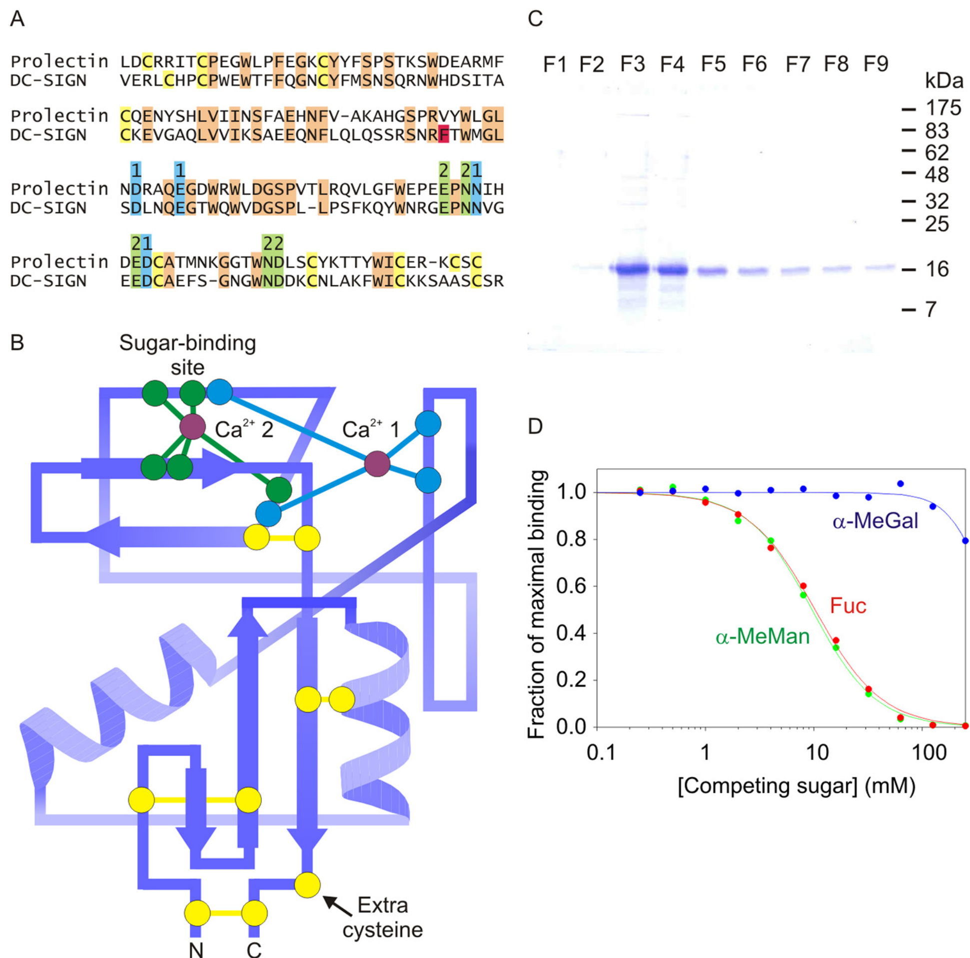


FIGURE 2. **Characterization of the CRD in prolectin.** *A*, sequence comparison between the CRDs of DC-SIGN and prolectin, with cysteine residues highlighted in *yellow*, ligands for Ca^{2+} 1 and 2 highlighted in *blue* and *green*, and other conserved residues *shaded orange*. Phe^{325} of DC-SIGN, which forms a critical part of the secondary binding site, is highlighted in *red*. *B*, diagram of the proposed topology of the CRD from prolectin, based on the structure of the CRD from DC-SIGN (Protein Data Base entry 1K9I). Conserved residues are highlighted in the same colors as in *A*. *C*, gel showing purification of the extracellular domain of prolectin expressed in *E. coli*. Aliquots of fractions eluted from a mannose-Sepharose affinity column with EDTA were run on the gel, which was stained with Coomassie Blue. *D*, examples of solid-phase binding competition assays using monosaccharides to inhibit binding of ^{125}I-Man-BSA (radiolabeled, mannose-conjugated bovine serum albumin) to the extracellular domain of prolectin immobilized in polystyrene wells.

column was constructed with bacterially expressed CRD immobilized on Affi-Gel 10 (Bio-Rad Laboratories). Monoclonal antibodies to phosphotyrosine were obtained from Amersham Biosciences (4G10) and BIOMOL International (PY20), and polyclonal antibody to Grb2 was purchased from Cell Signaling Technology. Alkaline phosphatase-conjugated protein A (Calbiochem) and goat anti-mouse IgG (Jackson ImmunoResearch), visualized with 5-bromo-4-chloro-3-indolyl nitroblue tetrazolium phosphatase substrate (Calbiochem), were used to counterstain blots.

Immunohistochemistry and Immunofluorescence—Formalin-fixed, paraffin-embedded tissue array slides of human normal organs were provided by SuperBioChips. The antibodies used were: mouse anti-Ki67, clone MIB-1 (1:80 dilution), mouse anti-CD68, clone KP1 (1:40), mouse anti-CD21 (1:33), and negative control mouse IgG1 (all Dako); affinity-purified rabbit anti-prolectin (10 μg/ml); and negative control normal rabbit IgG (gift from David Guiliano, Imperial College London). Slides were deparaffinized in xylene and rehydrated through a graded ethanol series. Heat-mediated antigen retrieval was performed using antigen unmasking solution (Vector Laboratories), except in the case of anti-CD21, where retrieval was performed using trypsin enzymatic antigen retrieval solution (Abcam). Sections were blocked with 10% normal goat serum (Vector Laboratories), and incubated for 90–120 min with primary antibody in 2% serum.

For immunofluorescence, sections were incubated with Alexa Fluor 488 goat anti-rabbit IgG and Alexa Fluor 594 goat anti-mouse IgG (Invitrogen) and mounted using Vectashield mounting medium with 4′,6-diamidino-2-phenylindole (Vector Laboratories). For immunohistochemistry, sections were incubated with biotinylated goat anti-rabbit IgG or anti-mouse IgG (Vector Laboratories), followed by quenching of endogenous peroxidase by 15-min incubation in 3% H_2O_2 in phosphate-buffered saline. Antigen was visualized by the peroxidase method using the Vectastain elite ABC and DAB substrate kits, and counterstained with Vector Hematoxylin QS (Vector Laboratories). Sections were dehydrated, cleared, and mounted with VectaMount permanent mounting medium (Vector Laboratories). Microscopy was performed on a Nikon Eclipse E400 microscope equipped with a DXM1200 digital camera. Images were captured with Lucia GF software, version 4.60, and were merged in Corel PhotoPaint 12.

RESULTS

The Prolectin Gene Encodes a Novel Receptor Containing a C-type CRD—A previously undescribed C-type CRD was identified in an entry denoted FLJ45910 protein (GenBank™ accession EAW84437) in the human genome annotation submitted by Celera Genomics. The domain contained key residues that typically form a Ca^{2+}-dependent sugar-binding site in C-type glycan-binding receptors (12). On further examination, the genomic region encoding this predicted gene was found to correspond in part to a gene recently given symbol *CLEC17A* (GeneID 388512) by the HUGO Gene Nomenclature Committee. The predicted protein from this interpretation of the genome data (UniProtKB/Swiss-Prot accession number Q6ZS10) lacks the C-terminal portion of the CRD.

Based on the predicted cDNA sequence from the Celera annotation, primers were designed for PCR amplification of a cDNA encoding the putative receptor, and receptor mRNA was

Downloaded from www.jbc.org by guest, on May 3, 2011

detected in libraries from multiple tissues. After this cDNA was cloned, the sequence of an IMAGE clone (13) that has an extended 5′ region was deposited in GenBank™ (GenBank™ accession number BC140848 for the cDNA, and UniProtKB/Swiss-Prot accession number B2RTX0 for the deduced amino acid sequence). Cloning with primers in this region confirmed the presence of a longer transcript which matched exactly the sequence of the IMAGE clone in the data base. A full-length cDNA, including this region, was used as a basis for further experiments.

Using the full-length cDNA as a guide, the complete prolectin gene structure, shown in Fig. 1*A*, was deduced. Comparison of the gene and cDNA sequences revealed that there must be an unusual GC splice donor site that is utilized instead of the nearby canonical GT sequence used in the Celera annotation. This splice results in a deletion of four amino acids in the actual sequence compared with the predicted sequence (Fig. 1*B*). The sequence flanking the GC splice donor site, AGGCAAGT, exactly matches the ideal sequence for binding to the U2-type spliceosome, which is believed to compensate for the absence of the canonical GT donor site (14). The length of the introns near the 3′ and 5′ ends of the gene, combined with the unusual splice site, are probably responsible for the fact that the prolectin cDNA was not correctly identified by any of the gene prediction algorithms.

The prolectin gene is located on the long arm of human chromosome 19, which contains a cluster of genes for several other receptors that utilize C-type CRDs, including the dendritic cell receptor DC-SIGN (Fig. 1*C*). However, the new gene is located roughly 7 mega bases away from this gene cluster.

The complete prolectin cDNA sequence encodes a type II transmembrane protein with an extended intracellular N-terminal domain and a C-terminal extracellular C-type CRD (Fig. 1*D*). The cytoplasmic domain contains repeated motifs that match patterns for binding to intracellular signaling molecules. There are at least three potential SH2-binding sites and eight potential SH3-binding sequences (15). Such a large cytoplasmic domain with so many potential interactions is unprecedented for a member of the C-type lectin family. These results suggest that the receptor has novel properties expected for a sugar-binding receptor with signaling functions.

TABLE 1

Summary of prolectin competition binding data

K_I values are shown relative to the value for αMe-Man run in parallel in each assay. The average K_I for αMe-Man was 9.9 ± 1.2 mM.

Ligand	Relative K_I
αMe-Man	1.0
αMe-Glc	3.2 ± 0.1
αMe-Gal	>30
Man	1.2 ± 0.1
GlcNAc	1.2 ± 0.1
Fuc	1.1 ± 0.1
Le^a	0.22 ± 0.01
Le^x	0.72 ± 0.08
Man_9	0.064 ± 0.04

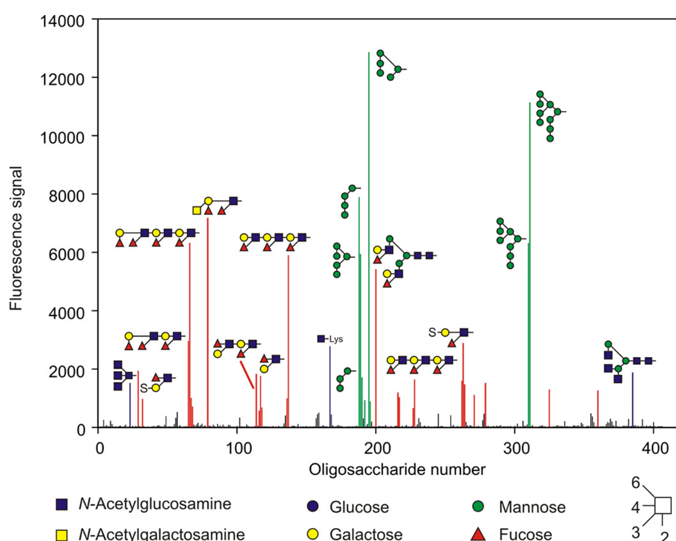


FIGURE 3. **Probing a glycan array with a tetrameric complex of Alexa 488-labeled streptavidin with biotin-tagged CRD from prolectin.** The protein concentration was 0.45 mg/ml. Glycans that are bound by the probe are *colored* based on the terminal residues present: *red* for fucose, *green* for mannose, and *blue* for *N*-acetylglucosamine. Structures of the ligands that give the strongest signals are indicated using *symbol nomenclature*. A complete list of all the glycans on the array is provided in supplemental Table S1.

The Extracellular Domain of Prolectin Binds Two Classes of Cell Surface Glycans—Based on the presence of a characteristic pattern of amino acids forming the conserved Ca^{2+}-binding site in C-type CRDs such as DC-SIGN, it was predicted that the protein encoded by this novel gene would bind mannose and related sugars (Fig. 2, *A* and *B*) (12, 16). The extracellular domain was therefore produced in a bacterial expression system and tested for binding to immobilized mannose. The protein was found to bind tightly to the resin and was eluted with EDTA (Fig. 2*C*). Binding was confirmed in a solid-phase assay, in which receptor extracellular domain immobilized in polystyrene wells was probed with iodinated, mannose-conjugated serum albumin (^{125}I-Man-BSA). Competition with monosaccharides revealed similar binding affinities for mannose, fucose, and *N*-acetylglucosamine, with much weaker binding to galactose (Fig. 2*D* and Table 1).

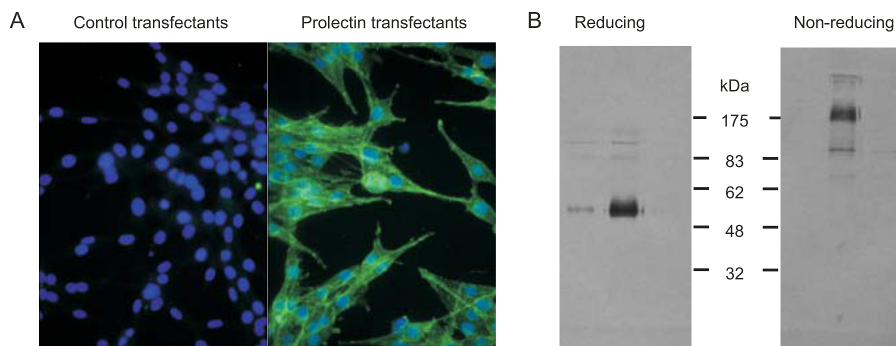
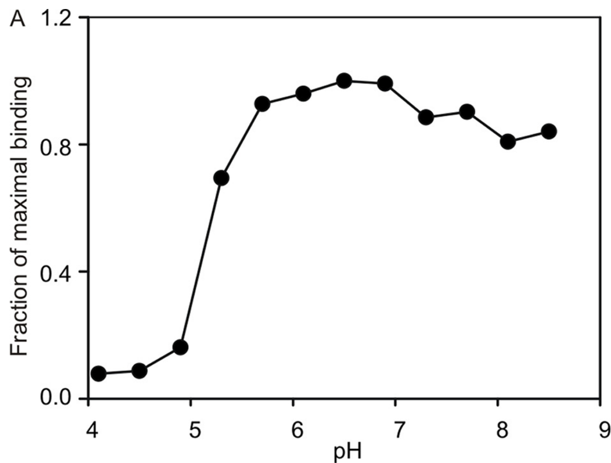


FIGURE 4. **Expression of prolectin in fibroblasts.** *A*, immunofluorescence of transfectants and control cells with anti-prolectin antibodies followed by Alexa 488-labeled secondary antibodies. Nuclei were stained with 4′,6-diamidino-2-phenylindole. *B*, gel electrophoresis of intact prolectin isolated from transfected fibroblasts. Aliquots of three peak fractions from mannose-Sepharose affinity column were run on gels in the presence and absence of reducing agent. Protein was detected by blotting the gel and probing with antibodies to prolectin followed by alkaline phosphatase-labeled protein A.

Downloaded from www.jbc.org by guest, on May 3, 2011



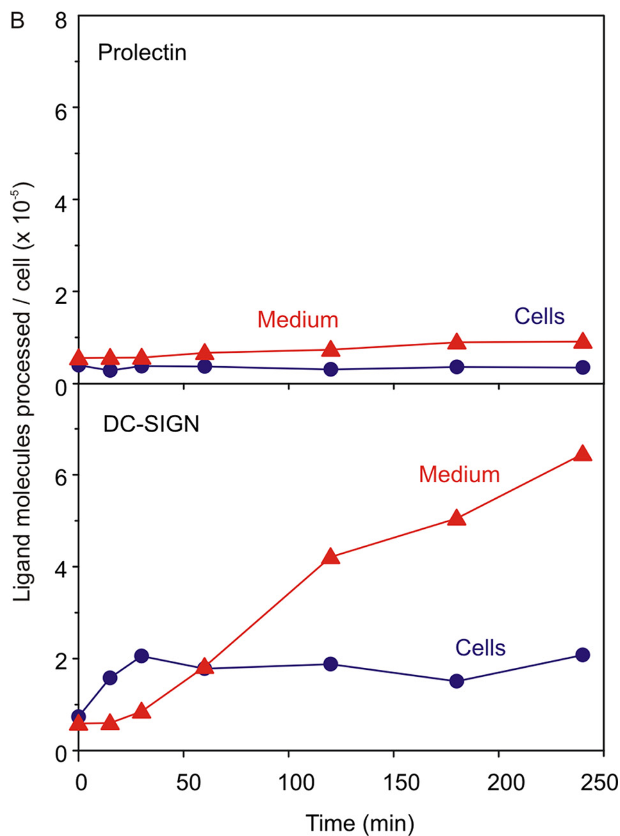


FIGURE 5. **Trafficking of prolectin expressed in fibroblasts.** *A*, pH dependence of ligand binding. Binding of ^{125}I-Man-BSA to immobilized extracellular domain of prolectin was assayed at various pHs in the presence of 2 mM Ca^{2+}. *B*, uptake and degradation of ^{125}I-Man-BSA by fibroblasts expressing prolectin or DC-SIGN. Degradation was measured as acid-soluble fragments appearing in the medium.

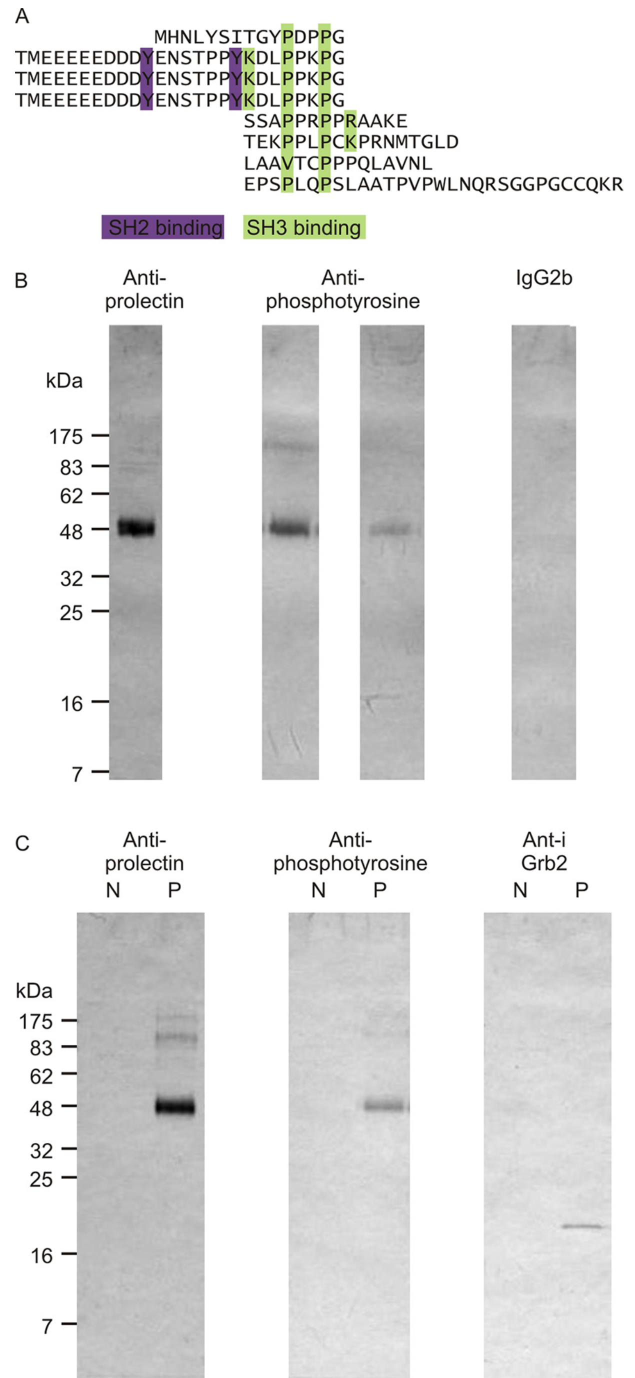


FIGURE 6. **Potential signaling interactions of prolectin.** *A*, potential binding motifs for SH2 and SH3 domains in the cytoplasmic domain of prolectin. *B*, prolectin phosphorylation. Aliquots of prolectin-containing fractions from mannose-Sepharose columns of extracts from transfected fibroblasts were separated on 17.5% SDS-polyacrylamide gels, which were blotted and probed with antibody to the CRD of prolectin, two different anti-phosphotyrosine monoclonal antibodies (*left*, 4G10; *right*, PY20) and a control mouse IgG2b antibody. *C*, cytoplasmic tail interactions. Aliquots of elution fractions from mannose-Sepharose columns of extracts from fibroblasts transfected with empty vector (*N*) and with vector containing the prolectin cDNA (*P*) were separated on 17.5% SDS-polyacrylamide gels, which were blotted and probed with antibody to the CRD of prolectin, anti-phosphotyrosine (monoclonal antibody 4G10), and affinity-purified polyclonal antibody to Grb2.

A more detailed analysis of sugar-binding specificity was undertaken by probing a glycan array containing more than 400 oligosaccharide ligands covalently immobilized on glass. CRD expressed with a C-terminal biotinylation tag was bound to fluorescently labeled streptavidin, providing a tetravalent complex that was screened for binding to the array. The ligands detected on the array fall largely into two groups, displaying either terminal mannose or terminal fucose residues (Fig. 3). Closer examination of the mannose-containing ligands indicates that the strongest signals are obtained with structures containing terminal Manα1–2Manα1–2Man sequences and that the fucose-containing ligands include terminal Lewis^a, Lewis^x, and Lewis^y structures. Probing of the array at lower

Downloaded from www.jbc.org by guest, on May 3, 2011

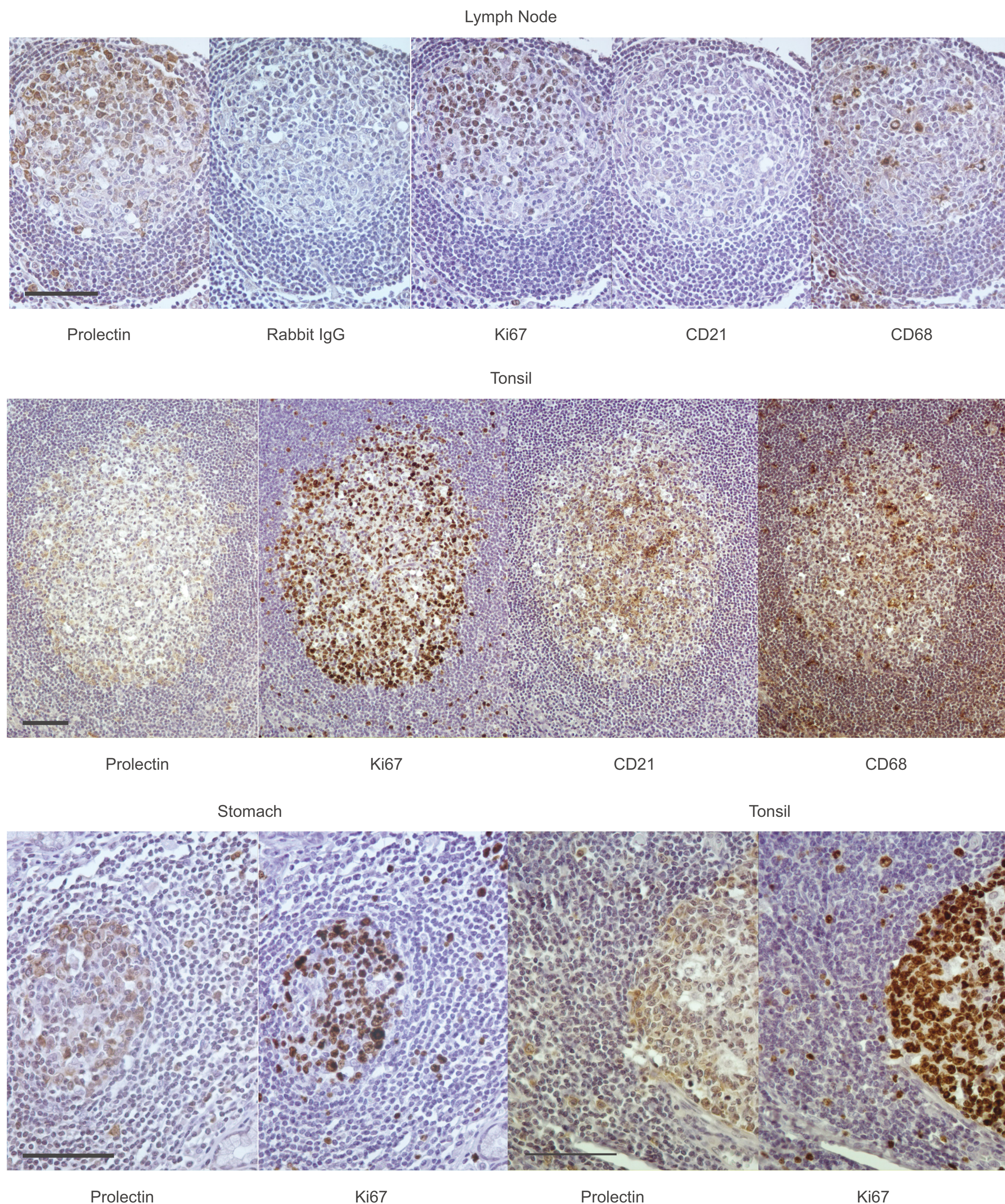


FIGURE 7. **Immunohistochemical localization of prolectin.** Germinal centers from multiple tissues were probed with polyclonal antibodies to prolectin, control rabbit IgG, or monoclonal antibodies specific for dividing cell nuclear antigen Ki67, dendritic cell marker CD21, and macrophage marker CD68. Antigens were visualized with peroxidase immunochemistry. *Scale bars*, 100 μm.

protein concentration resulted in reduction of all the signals, indicating that no one of these categories of ligand binds with dramatically higher affinity than others. The observed binding to both high mannose and Lewis-type structures was also confirmed in competition assays (Table 1). These assays indicate that the Lewis^a trisaccharide is a better ligand for prolectin than is Lewis^x, suggesting that the apparent preference for Lewis^x-containing structures on the array is a result of the

Downloaded from www.jbc.org by guest, on May 3, 2011

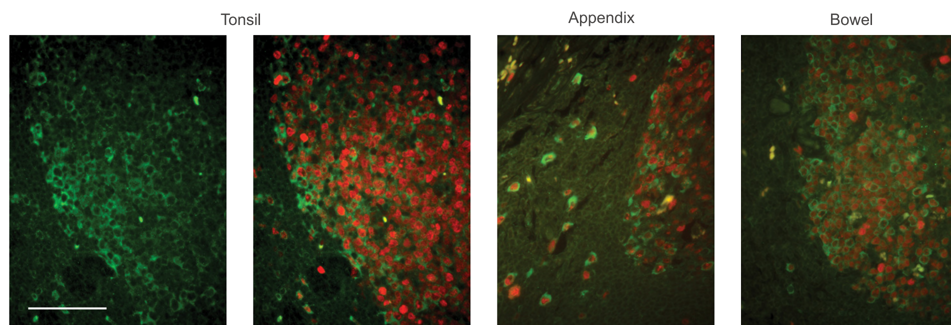


FIGURE 8. **Comparison of prolectin and Ki67 antigen distribution.** Germinal centers were stained with antibody to prolectin and counterstained with Alexa 488 secondary antibody (*green*). In the three images on the *right*, sections were also stained with Ki67 antibody and counterstained with Alexa 594 secondary antibody (*red*). *Scale bar*, 100 μm.

disposition of these structures, often in multiple copies, on larger oligosaccharides.

The profile of results is similar to that obtained for the dendritic cell receptor DC-SIGN, but there are significant differences between the preferred ligands for the two proteins (16). Binding of oligosaccharides to C-type CRDs usually involves docking of a monosaccharide into the primary binding site at a conserved Ca^{2+}, with enhanced affinity for the oligosaccharide resulting from secondary contacts between other sugars in the oligosaccharide and residues nearby on the protein surface (12, 16). A phenylalanine residue in DC-SIGN that makes key contacts with portions of high mannose oligosaccharides and a valine residue that contributes to the specificity of DC-SIGN for Lewis^x and Lewis^a structures are not conserved in prolectin, suggesting that the secondary binding sites in the two receptors must be different. This conclusion is consistent with the observed differences in selectivity for different ligands within broadly similar classes.

Prolectin Is Expressed at the Cell Surface—The properties of prolectin were examined by stable expression of a full-length cDNA in Rat-6 fibroblasts, cells that display no detectable surface glycan-binding receptors but which have been shown to support activity of multiple exogenous receptors containing C-type CRDs (11, 16, 17). Immunofluorescence staining revealed that prolectin appears on the cell surface of the transfectants (Fig. 4*A*), and receptor isolated from the fibroblasts was found to form disulfide-linked oligomers with molecular mass in excess of 175 kDa (Fig. 4*B*). Based on the previously characterized pattern of disulfide bonds within C-type CRDs (18, 19), a single free cysteine residue would be present in the CRD (Fig. 2*B*). Interchain disulfide bonding of this cysteine could lead to dimer formation, but the larger size of the oligomers observed would require formation of additional interchain disulfide bonds involving one of the cysteine residues in the transmembrane domain of the receptor. Dimerization of dimers brought about in this way would lead to tetramer formation, which would be consistent with the observed size of the disulfide-linked oligomer observed on gels.

Oligomer formation is a common feature of glycan-binding receptors and can be an important determinant of the way that they interact with glycans on cell surfaces (12, 20, 21). Many receptors form dimers, and DC-SIGN forms tetramers, but they are noncovalent and the neck sequence that supports oligomer formation in DC-SIGN is unrelated to the sequence of the neck in prolectin (22).

Because many oligomeric type II receptors that contain C-type CRDs mediate endocytosis (11, 16, 17), it was of interest to see if prolectin supports endocytic activity. The pH profile of ligand binding in the solid-phase assay showed that ligand release occurred at low pH, but that half-maximal release occurred at pH 5.2 (Fig. 5*A*). This value is substantially lower than the typical value for recycling endocytic receptors such as DC-SIGN and the hepatic asialoglycoprotein receptor (16, 23). When ^{125}I-Man-BSA was incubated with the transfected fibroblasts, no uptake and processing of ligand was observed, providing a sharp contrast with DC-SIGN transfectants assayed in parallel (Fig. 5*B*).

The Cytoplasmic Domain of Prolectin Communicates with Signaling Molecules—Although prolectin does not support endocytosis, examination of the cytoplasmic domain suggested the alternative possibility that it has signaling capacity, because of the presence of numerous sequences predicted to bind SH2 and SH3 domains (Fig. 6*A*). In the oligomeric protein, these sequences would form large clusters of potential binding sites for adapter molecules that could link the receptor to intracellular signaling pathways. The SH2-binding sequences contain tyrosine residues that would be expected to be targets for tyrosine kinases and antibody probing of blots of prolectin purified from the fibroblasts confirmed the presence of phosphotyrosine (Fig. 6*B*).

The sequence context of the phosphorylated tyrosine residues suggests potential interactions with specific classes of cytoplasmic signaling molecules (24). In particular, the ubiquitous adapter protein Grb2 contains an SH2 domain that binds the target sequence Y*X*N*X* (25). Interestingly, Grb2 also contains class I SH3 domains that interact with the target SH3-binding motifs identified in prolectin. Consistent with these predictions, antibody blotting of the receptor-containing fractions from the mannose-Sepharose affinity column demonstrated that Grb2 co-purifies with prolectin (Fig. 6*C*), confirming that prolectin interacts with intracellular signaling machinery either directly or indirectly.

Prolectin Is Expressed on Dividing B Cells of Germinal Centers—Immunohistochemistry using the affinity-purified polyclonal rabbit antibodies was used to screen an array of human tissues to define sites of expression. The results revealed a very restricted pattern of staining, with the major site of expression being germinal centers in various tissues, including lymph nodes, tonsils, stomach, intestine, appendix, and spleen (Fig. 7). Antibodies selective for follicular dendritic cells or tingible body macrophages found in the light zone of the germinal centers do not co-localize with prolectin. Thus, unlike other C-type lectins such as DC-SIGN and the mannose receptor, prolectin is not found on dendritic cells or macrophages. In contrast, Ki67 antigen found in nuclei of dividing B cells shows extensive

Downloaded from www.jbc.org by guest, on May 3, 2011

overlap with the prolectin staining, suggesting a novel localization of this receptor in B cells.

Co-localization of prolectin with the dividing B cell marker Ki67 was confirmed using immunofluorescence (Fig. 8). In germinal centers from multiple tissues, essentially all prolectin-expressing cells are also Ki67-positive. Based on their location and reaction with the anti-Ki67 antibody, the clustered, doubly stained cells in the dark zone appear to be centroblasts. Prolectin expression thus defines a sub-population of dividing B cells of the dark zone. In the surrounding mantle zone, the occasional Ki67-positive cells have a similar morphology to the prolectin-positive cells in the dark zone and all of these cells co-express prolectin.

DISCUSSION

Although prolectin has some similarity to other glycan-binding receptors containing C-type CRDs, it has several properties that distinguish it from other known members of this group. Importantly, the fact that prolectin does not display endocytic activity is consistent with its expression in B cells rather than macrophages or dendritic cells. This pattern of expression and lack of endocytic activity makes it unlikely that prolectin functions as a primary recognition receptor in the innate immune response, a role that is commonly ascribed to DC-SIGN, langerin, the mannose receptor, and other receptors that contain C-type CRDs (1, 26–28). Similarly, in contrast to the selectin cell adhesion molecules, prolectin bears multiple signaling motifs in the cytoplasmic domain, so that it can interact directly with signaling pathways within lymphocytes, although it may also have a role in cell adhesion. Thus, prolectin does not readily fall into any of the previously described categories of C-type lectins, and it does not have any obvious paralogs in mammalian genomes.

Despite these differences, the glycan array results suggest that that there is likely to be overlap in potential ligands for prolectin and DC-SIGN, because both receptors display a dual specificity for mannose- and fucose-terminated glycans. Common types of ligands would include surface glycans on other cells bearing Lewis^a, Lewis^x, and Lewis^y epitopes (16, 29). It has been proposed that these structures are presented to DC-SIGN on intercellular adhesion molecule 3 (ICAM-3) on T cells, leading to adhesion between the T cells and dendritic cells (30). By analogy, one function of prolectin may be to participate in interaction of the B cells on which it is expressed with T cells or other cells in the germinal center. However, the potential ability of prolectin to initiate signaling in response to a glycan ligand indicates that it probably has additional roles in communication between B cells and other cells in germinal centers.

The pattern of expression of prolectin within the germinal centers may provide some additional clues about its potential functions. Following somatic mutation of antibody genes in centroblasts of the dark zone, centrocytes are known to migrate to the light zone where selection of cells with enhanced affinity for antigen occurs and cells with dysfunctional immunoglobulin genes undergo apoptosis (31). The disposition of clusters containing many of the prolectin-positive cells near the edge of the dark zone, and the presence of individual cells in the adjacent regions of the mantle zone suggests that cells expressing prolectin might be in transit into or out of the germinal center and that the receptor may play a role in migration of cells between these compartments during antibody affinity maturation.

Acknowledgments—We thank David Guiliano, Niki Gounaris, and Murray Selkirk of the Division of Molecular and Cellular Biology, Imperial College, for providing access to the fluorescence microscope and David Smith, Department of Biochemistry, Emory University School of Medicine, for performing the glycan array analysis.

REFERENCES

1. Weis, W. I., Taylor, M. E., and Drickamer, K. (1998) *Immunol. Rev.* **163,** 19–34
2. van Kooyk, Y., and Rabinovich, G. A. (2008) *Nat. Immunol.* **9,** 593–601
3. Vestweber, D., and Blanks, J. E. (1999) *Physiol. Rev.* **79,** 181–213
4. Rosen, S. D. (2004) *Annu. Rev. Immunol.* **22,** 129–156
5. Crocker, P. R., Paulson, J. C., and Varki, A. (2007) *Nat. Rev. Immunol.* **7,** 255–266
6. Drickamer, K., and Taylor, M. E. (2003) *Methods Enzymol.* **362,** 560–567
7. Mitchell, D. A., Fadden, A. J., and Drickamer, K. (2001) *J. Biol. Chem.* **276,** 28939–28945
8. Fornstedt, N., and Porath, J. (1975) *FEBS Lett.* **57,** 187–191
9. Laemmli, U. K. (1970) *Nature* **227,** 680–685
10. Powlesland, A. S., Ward, E. M., Sadhu, S. K., Guo, Y., Taylor, M. E., and Drickamer, K. (2006) *J. Biol. Chem.* **281,** 20440–20449
11. Stambach, N. S., and Taylor, M. E. (2003) *Glycobiology* **13,** 401–410
12. Weis, W. I., and Drickamer, K. (1996) *Annu. Rev. Biochem.* **65,** 441–473
13. Lennon, G., Auffray, C., Polymeropoulos, M., and Soares, M. B. (1996) *Genomics* **33,** 151–152
14. Thanaraj, T. A., and Clark, F. (2001) *Nucleic Acids Res.* **29,** 2581–2593
15. Puntervoll, P., Linding, R., Gemünd, C., Chabanis-Davidson, S., Mattingsdal, M., Cameron, S., Martin, D. M., Ausiello, G., Brannetti, B., Costantini, A., Ferrè, F., Maselli, V., Via, A., Cesareni, G., Diella, F., Superti-Furga, G., Wyrwicz, L., Ramu, C., McGuigan, C., Gudavalli, R., Letunic, I., Bork, P., Rychlewski, L., Küster, B., Helmer-Citterich, M., Hunter, W. N., Aasland, R., and Gibson, T. J. (2003) *Nucleic Acids Res.* **31,** 3625–3630
16. Guo, Y., Feinberg, H., Conroy, E., Mitchell, D. A., Alvarez, R., Blixt, O., Taylor, M. E., Weis, W. I., and Drickamer, K. (2004) *Nat. Struct. Mol. Biol.* **11,** 591–598
17. Coombs, P. J., Graham, S. A., Drickamer, K., and Taylor, M. E. (2005) *J. Biol. Chem.* **280,** 22993–22999
18. Feinberg, H., Mitchell, D. A., Drickamer, K., and Weis, W. I. (2001) *Science* **294,** 2163–2166
19. Wurzburg, B. A., Tarchevskaya, S. S., and Jardetzky, T. S. (2006) *Structure* **14,** 1049–1058
20. Drickamer, K. (1999) *Curr. Opin. Struct. Biol.* **9,** 585–590
21. Dam, T. K., and Brewer, C. F. (2008) *Biochemistry* **47,** 8470–8476
22. Feinberg, H., Guo, Y., Mitchell, D. A., Drickamer, K., and Weis, W. I. (2005) *J. Biol. Chem.* **280,** 1327–1335
23. Wragg, S., and Drickamer, K. (1999) *J. Biol. Chem.* **274,** 35400–35406
24. Pawson, T., and Scott, J. D. (1997) *Science* **278,** 2075–2080
25. Chardin, P., Cussac, D., Maignan, S., and Ducruix, A. (1995) *FEBS Lett.* **369,** 47–51
26. van Kooyk, Y., and Geijtenbeek, T. B. H. (2003) *Nat. Rev. Immunol.* **3,** 697–709
27. de Witte, L., Nabatov, A., Pion, M., Fluitsma, D., de Jong, M. A., de Gruijl, T., Piguet, V., van Kooyk, Y., and Geijtenbeek, T. B. (2007) *Nat. Med.* **13,** 367–371
28. Taylor, P. R., Gordon, S., and Martinez-Pomares, L. (2005) *Trends Immunol.* **26,** 104–110
29. Appelmelk, B. J., van Die, I., van Vliet, S. J., Vandenbroucke-Grauls, C. M., Geijtenbeek, T. B., and van Kooyk, Y. (2003) *J. Immunol.* **170,** 1635–1639
30. Geijtenbeek, T. B., Torensma, R., van Vliet, S. J., van Duijnhoven, G. C., Adema, G. J., van Kooyk, Y., and Figdor, C. G. (2000) *Cell* **100,** 575–585
31. Allen, C. D., Okada, T., and Cyster, J. G. (2007) *Immunity* **27,** 190–202

Downloaded from www.jbc.org by guest, on May 3, 2011

Supplementary Information for

PROLECTIN: A GLYCAN-BINDING RECEPTOR ON DIVIDING B CELLS IN GERMINAL CENTERS

Sarah A. Graham, Sabine A. F. Jégouzo, Sheng Yan, Alex S. Powlesland, Jacob P. Brady, Maureen E. Taylor, and Kurt Drickamer

SUPPLEMENTARY TABLE 1
Oligosaccharides on glycan array

Glycan number	Glycan Structure
1	Neu5Acα2-8Neu5Acβ-Sp17
2	Neu5Acα2-8Neu5Acα2-8Neu5Acβ-Sp8
3	Neu5Gcβ2-6Gαlβ1-4GlcNAc-Sp8
4	Galβ1-3GlcNAcβ1-2Manα1-3(Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp19
5	Galα-Sp8
6	Glcα-Sp8
7	Manα-Sp8
8	GalNAcα-Sp8
9	Fucα-Sp8
10	Fucα-Sp9
11	Rha-Sp8
12	Neu5Acα-Sp8
13	Neu5Acα-Sp11
14	Neu5Acβ-Sp8
15	Galβ-Sp8
16	Glcβ-Sp8
17	Manβ-Sp8
18	GalNAcβ-Sp8
19	GlcNAcβ-Sp0
20	GlcNAcβ-Sp8
21	GlcN(Gc)β-Sp8
22	Galβ1-4GlcNAcβ1-3(Galβ1-4GlcNAcβ1-6)GalNAcα-Sp8
23	GlcNAcβ1-3(GlcNAcβ1-4)(GlcNAcβ1-6)GlcNAc-Sp8
24	[3OSO3][6OSO3]Galβ1-4[6OSO3]GlcNAcβ-Sp0
25	[3OSO3][6OSO3]Galβ1-4GlcNAcβ-Sp0
26	[3OSO3]Galβ1-4Glcβ-Sp8
27	[3OSO3]Galβ1-4[6OSO3]Glcβ-Sp0
28	[3OSO3]Galβ1-4[6OSO3]Glcβ-Sp8
29	[3OSO3]Galβ1-3(Fucα1-4)GlcNAcβ-Sp8
30	[3OSO3]Galβ1-3GalNAcα-Sp8
31	[3OSO3]Galβ1-3GlcNAcβ-Sp8
32	[3OSO3]Galβ1-4(Fucα1-3)GlcNAcβ-Sp8
33	[3OSO3]Galβ1-4[6OSO3]GlcNAcβ-Sp8
34	[3OSO3]Galβ1-4GlcNAcβ-Sp0
35	[3OSO3]Galβ1-4GlcNAcβ-Sp8
36	[3OSO3]Galβ-Sp8
37	[4OSO3][6OSO3]Galβ1-4GlcNAcβ-Sp0
38	[4OSO3]Galβ1-4GlcNAcβ-Sp8
39	6-H2PO3Manα-Sp8
40	[6OSO3]Galβ1-4Glcβ-Sp0
41	[6OSO3]Galβ1-4Glcβ-Sp8
42	[6OSO3]Galβ1-4GlcNAcβ-Sp8

Downloaded from www.jbc.org by guest, on May 3, 2011

43	[6OSO3]Galβ1-4[6OSO3]Glcβ-Sp8
44	Neu5Acα2-3[6OSO3]Galβ1-4GlcNAcβ-Sp8
45	[6OSO3]GlcNAcβ-Sp8
46	Neu5Ac(9Ac)α-Sp8
47	Neu5Ac(9Ac)α2-6Galβ1-4GlcNAcβ-Sp8
48	Manα1-3(Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp13
49	GlcNAcβ1-2Manα1-3(GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp13
50	Galβ1-4GlcNAcβ1-2Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
51	Galβ1-4GlcNAcβ1-2Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp13
52	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
53	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp13
54	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp8
55	Fucα1-2Galβ1-3GalNAcβ1-3Galα-Sp9
56	Fucα1-2Galβ1-3GalNAcβ1-3Galα1-4Galβ1-4Glcβ-Sp9
57	Fucα1-2Galβ1-3(Fucα1-4)GlcNAcβ-Sp8
58	Fucα1-2Galβ1-3GalNAcα-Sp8
59	Fucα1-2Galβ1-3GalNAcβ1-4(Neu5Acα2-3)Galβ1-4Glcβ-Sp0
60	Fucα1-2Galβ1-3GalNAcβ1-4(Neu5Acα2-3)Galβ1-4Glcβ-Sp9
61	Fucα1-2Galβ1-3GlcNAcβ1-3Galβ1-4Glcβ-Sp10
62	Fucα1-2Galβ1-3GlcNAcβ1-3Galβ1-4Glcβ-Sp8
63	Fucα1-2Galβ1-3GlcNAcβ-Sp0
64	Fucα1-2Galβ1-3GlcNAcβ-Sp8
65	Fucα1-2Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
66	Fucα1-2Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
67	Fucα1-2Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
68	Fucα1-2Galβ1-4(Fucα1-3)GlcNAcβ-Sp8
69	Fucα1-2Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
70	Fucα1-2Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
71	Fucα1-2Galβ1-4GlcNAcβ-Sp0
72	Fucα1-2Galβ1-4GlcNAcβ-Sp8
73	Fucα1-2Galβ1-4Glcβ-Sp0
74	Fucα1-2Galβ-Sp8
75	Fucα1-3GlcNAcβ-Sp8
76	Fucα1-4GlcNAcβ-Sp8
77	Fucβ1-3GlcNAcβ-Sp8
78	GalNAcα1-3(Fucα1-2)Galβ1-3GlcNAcβ-Sp0
79	GalNAcα1-3(Fucα1-2)Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
80	GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ-Sp0
81	GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ-Sp8
82	GalNAcα1-3(Fucα1-2)Galβ1-4Glcβ-Sp0
83	GalNAcα1-3(Fucα1-2)Galβ-Sp8
84	GalNAcα1-3GalNAcβ-Sp8
85	GalNAcα1-3Galβ-Sp8
86	GalNAcα1-4(Fucα1-2)Galβ1-4GlcNAcβ-Sp8
87	GalNAcβ1-3GalNAcα-Sp8
88	GalNAcβ1-3(Fucα1-2)Galβ-Sp8
89	GalNAcβ1-3Galα1-4Galβ1-4GlcNAcβ-Sp0
90	GalNAcβ1-4(Fucα1-3)GlcNAcβ-Sp0
91	GalNAcβ1-4GlcNAcβ-Sp0

Downloaded from www.jbc.org by guest, on May 3, 2011

92	GalNAcβ1-4GlcNAcβ-Sp8
93	Galα1-2Galβ-Sp8
94	Galα1-3(Fucα1-2)Galβ1-3GlcNAcβ-Sp0
95	Galα1-3(Fucα1-2)Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
96	Galα1-3(Fucα1-2)Galβ1-4GlcNAc-Sp0
97	Galα1-3(Fucα1-2)Galβ1-4Glcβ-Sp0
98	Galα1-3(Fucα1-2)Galβ-Sp8
99	Galα1-3(Galα1-4)Galβ1-4GlcNAcβ-Sp8
100	Galα1-3GalNAcα-Sp8
101	Galα1-3GalNAcβ-Sp8
102	1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp8
103	Galα1-3Galβ1-3GlcNAcβ-Sp0
104	Galα1-3Galβ1-4GlcNAcβ-Sp8
105	Galα1-3Galβ1-4Glcβ-Sp0
106	Galα1-3Galβ-Sp8
107	Galα1-4(Fucα1-2)Galβ1-4GlcNAcβ-Sp8
108	Galα1-4Galβ1-4GlcNAcβ-Sp0
109	Galα1-4Galβ1-4GlcNAcβ-Sp8
110	Galα1-4Galβ1-4Glcβ-Sp0
111	Galα1-4GlcNAcβ-Sp8
112	Galα1-6Glcβ-Sp8
113	Galβ1-2Galβ-Sp8
114	Galβ1-3(Fucα1-4)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
115	Galβ1-3(Fucα1-4)GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
116	Galβ1-3(Fucα1-4)GlcNAcβ-Sp0
117	Galβ1-3(Fucα1-4)GlcNAc-Sp8
118	Galβ1-3(Fucα1-4)GlcNAcβ-Sp8
119	Galβ1-3(Galβ1-4GlcNAcβ1-6)GalNAcα-Sp8
120	Galβ1-3(GlcNAcβ1-6)GalNAcα-Sp8
121	Galβ1-3(Neu5Acα2-6)GalNAcα-Sp8
122	Galβ1-3(Neu5Acβ2-6)GalNAcα-Sp8
123	Galβ1-3(Neu5Acα2-6)GlcNAcβ1-4Galβ1-4Glcβ-Sp10
124	Galβ1-3GalNAcα-Sp8
125	Galβ1-3GalNAcβ-Sp8
126	Galβ1-3GalNAcβ1-3Galα1-4Galβ1-4Glcβ-Sp0
127	Galβ1-3GalNAcβ1-4(Neu5Acα2-3)Galβ1-4Glcβ-Sp0
128	Galβ1-3GalNAcβ1-4Galβ1-4Glcβ-Sp8
129	Galβ1-3Galβ-Sp8
130	Galβ1-3GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
131	Galβ1-3GlcNAcβ1-3Galβ1-4Glcβ-Sp10
132	Galβ1-3GlcNAcβ-Sp0
133	Galβ1-3GlcNAcβ-Sp8
134	Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
135	Galβ1-4(Fucα1-3)GlcNAcβ-Sp8
136	Galβ1-4(Fucα1-3)GlcNAcβ1-4Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
137	Galβ1-4(Fucα1-3)GlcNAcβ1-4Galβ1-4(Fucα1-3)GlcNAcβ1-4Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
138	Galβ1-4[6OSO3]Glcβ-Sp0
139	Galβ1-4[6OSO3]Glcβ-Sp8
140	Galβ1-4GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ-Sp8
141	Galβ1-4GalNAcβ1-3(Fucα1-2)Galβ1-4GlcNAcβ-Sp8
142	Neu5Acα2-3Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-3Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
143	Galβ1-4GlcNAcβ1-3GalNAcα-Sp8
144	Galβ1-4GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
145	Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0

Downloaded from www.jbc.org by guest, on May 3, 2011

146	Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
147	Galβ1-4GlcNAcβ1-3Galβ1-4Glcβ-Sp0
148	Galβ1-4GlcNAcβ1-3Galβ1-4Glcβ-Sp8
149	Galβ1-4GlcNAcβ1-6(Galβ1-3)GalNAcα-Sp8
150	Galβ1-4GlcNAcβ1-6GalNAcα-Sp8
151	Galβ1-4GlcNAcβ-Sp0
152	Galβ1-4GlcNAcβ-Sp8
153	Galβ1-4Glcβ-Sp0
154	Galβ1-4Glcβ-Sp8
155	GlcNAcα1-3Galβ1-4GlcNAcβ-Sp8
156	GlcNAcα1-6Galβ1-4GlcNAcβ-Sp8
157	GlcNAcβ1-2Galβ1-3GalNAcα-Sp8
158	GlcNAcβ1-3(GlcNAcβ1-6)GalNAcα-Sp8
159	GlcNAcβ1-3(GlcNAcβ1-6)Galβ1-4GlcNAcβ-Sp8
160	GlcNAcβ1-3GalNAcα-Sp8
161	GlcNAcβ1-3Galβ-Sp8
162	GlcNAcβ1-3Galβ1-3GalNAcα-Sp8
163	GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
164	GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp8
165	GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
166	GlcNAcβ1-3Galβ1-4Glcβ-Sp0
167	GlcNAcβ1-4-MDPLys
168	GlcNAcβ1-4(GlcNAcβ1-6)GalNAcα-Sp8
169	GlcNAcβ1-4Galβ1-4GlcNAcβ-Sp8
170	(GlcNAcβ1-4)6β-Sp8
171	(GlcNAcβ1-4)5β-Sp8
172	GlcNAcβ1-4GlcNAcβ1-4GlcNAcβ-Sp8
173	GlcNAcβ1-6(Galβ1-3)GalNAcα-Sp8
174	GlcNAcβ1-6GalNAcα-Sp8
175	GlcNAcβ1-6Galβ1-4GlcNAcβ-Sp8
176	Glcα1-4Glcβ-Sp8
177	Glcα1-4Glcα-Sp8
178	Glcα1-6Glcα1-6Glcβ-Sp8
179	Glcβ1-4Glcβ-Sp8
180	Glcβ1-6Glcβ-Sp8
181	G-ol-Sp8
182	GlcAa-Sp8
183	GlcAβ-Sp8
184	GlcAβ1-3Galβ-Sp8
185	GlcAβ1-6Galβ-Sp8
186	KDNa2-3Galβ1-3GlcNAcβ-Sp0
187	KDNa2-3Galβ1-4GlcNAcβ-Sp0
188	Manα1-2Manα1-2Manα1-3Manα-Sp9
189	Manα1-2Manα1-3(Manα1-2Manα1-6)Manα-Sp9
190	Manα1-2Manα1-3Manα-Sp9
191	Manα1-6(Manα1-2Manα1-3)Manα1-6(Manα1-2Manα1-3)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
192	Manα1-2Manα1-6(Manα1-3)Manα1-6(Manα1-2Manα1-2Manα1-3)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
193	Manα1-2Manα1-2Manα1-3(Manα1-2Manα1-3(Manα1-2Manα1-6)Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
194	Manα1-3(Manα1-6)Manα-Sp9
195	Manα1-3(Manα1-2Manα1-2Manα1-6)Manα-Sp9
196	Manα1-6(Manα1-3)Manα1-6(Manα1-2Manα1-3)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
197	Manα1-6(Manα1-3)Manα1-6(Manα1-3)Manβ1-4GlcNAcβ1-4 GlcNAcβ-Sp12

Downloaded from www.jbc.org by guest, on May 3, 2011

198	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-3Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
199	Manβ1-4GlcNAcβ-Sp0
200	Fucα1-3(Galβ1-4)GlcNAcβ1-2Manα1-3(Fucα1-3(Galβ1-4)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
201	Neu5Acα2-3Galβ1-3GalNAcα-Sp8
202	Neu5Acα2-8Neu5Acα2-8Neu5Acα2-8Neu5Acα2-3(GalNAcβ1-4)Galβ1-4Glcβ-Sp0
203	Neu5Acα2-8Neu5Acα2-8Neu5Acα2-3(GalNAcβ1-4)Galβ1-4Glcβ-Sp0
204	Neu5Acα2-8Neu5Acα2-8Neu5Acα2-3Galβ1-4Glcβ-Sp0
205	Neu5Acα2-8Neu5Acα2-3(GalNAcβ1-4)Galβ1-4Glcβ-Sp0
206	Neu5Acα2-8Neu5Acα2-8Neu5Aca-Sp8
207	Neu5Acα2-3(6-O-Su)Galβ1-4(Fucα1-3)GlcNAcβ-Sp8
208	Neu5Acα2-3(GalNAcβ1-4)Galβ1-4GlcNAcβ-Sp0
209	Neu5Acα2-3(GalNAcβ1-4)Galβ1-4GlcNAcβ-Sp8
210	Neu5Acα2-3(GalNAcβ1-4)Galβ1-4Glcβ-Sp0
211	Neu5Acα2-3(Neu5Acα2-3Galβ1-3GalNAcβ1-4)Galβ1-4Glcβ-Sp0
212	Neu5Acα2-3(Neu5Acα2-6)GalNAcα-Sp8
213	Neu5Acα2-3GalNAcα-Sp8
214	Neu5Acα2-3GalNAcβ1-4GlcNAcβ-Sp0
215	Neu5Acα2-3Galβ1-3[6OSO3]GlcNAc-Sp8
216	Neu5Acα2-3Galβ1-3(Fucα1-4)GlcNAcβ-Sp8
217	Neu5Acα2-3Galβ1-3(Fucα1-4)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
218	Neu5Acα2-3Galβ1-3(Neu5Acα2-3Galβ1-4)GlcNAcβ-Sp8
219	Neu5Acα2-3Galβ1-3[6OSO3]GalNAcα-Sp8
220	Neu5Acα2-3Galβ1-3(Neu5Acα2-6)GalNAcα-Sp8
221	Neu5Acα2-3Galβ-Sp8
222	Neu5Acα2-3Galβ1-3GalNAcβ1-3Galα1-4Galβ1-4Glcβ-Sp0
223	Neu5Acα2-3Galβ1-3GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
224	Neu5Acα2-3Galβ1-3GlcNAcβ-Sp0
225	Neu5Acα2-3Galβ1-3GlcNAcβ-Sp8
226	Neu5Acα2-3Galβ1-4[6OSO3]GlcNAcβ-Sp8
227	Neu5Acα2-3Galβ1-4(Fucα1-3)[6OSO3]GlcNAcβ-Sp8
228	Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
229	Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
230	Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp8
231	Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ-Sp8
232	Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp8
233	Neu5Acα2-3Galβ1-4GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAc-Sp0
234	Neu5Acα2-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
235	Neu5Acα2-3Galβ1-4GlcNAcβ-Sp0
236	Neu5Acα2-3Galβ1-4GlcNAcβ-Sp8
237	Neu5Acα2-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
238	Neu5Acα2-3Galβ1-4Glcβ-Sp0
239	Neu5Acα2-3Galβ1-4Glcβ-Sp8
240	Neu5Acα2-6GalNAcα-Sp8
241	Neu5Acα2-6GalNAcβ1-4GlcNAcβ-Sp0
242	Neu5Acα2-6Galβ1-4[6OSO3]GlcNAcβ-Sp8
243	Neu5Acα2-6Galβ1-4GlcNAcβ-Sp0
244	Neu5Acα2-6Galβ1-4GlcNAcβ-Sp8
245	Neu5Acα2-6Galβ1-4GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
246	Neu5Acα2-6Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
247	Neu5Acα2-6Galβ1-4Glcβ-Sp0
248	Neu5Acα2-6Galβ1-4Glcβ-Sp8
249	Neu5Acα2-6Galβ-Sp8

Downloaded from www.jbc.org by guest, on May 3, 2011

250	Neu5Acα2-8Neu5Aca-Sp8
251	Neu5Acα2Acα2-8Neu5Acα2-3Galβ1-4Glcβ-Sp0
252	Neu5Acβ2-6GalNAcα-Sp8
253	Neu5Acβ2-6Galβ1-4GlcNAcβ-Sp8
254	Neu5Gca2-3Galβ1-3(Fucα1-4)GlcNAcβ-Sp0
255	Neu5Gca2-3Galβ1-3GlcNAcβ-Sp0
256	Neu5Gca2-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
257	Neu5Gca2-3Galβ1-4GlcNAcβ-Sp0
258	Neu5Gca2-3Galβ1-4Glcβ-Sp0
259	Neu5Gca2-6GalNAcα-Sp0
260	Neu5Gca2-6Galβ1-4GlcNAcβ-Sp0
261	Neu5Gca-Sp8
262	[3OSO3]Galβ1-4(Fucα1-3)[6OSO3]Glc-Sp0
263	[3OSO3]Galβ1-4(Fucα1-3)Glc-Sp0
264	[3OSO3]Galβ1-4(Fucα1-3)[6OSO3]GlcNAc-Sp8
265	[3OSO3]Galβ1-4(Fucα1-3)GlcNAc-Sp0
266	Fucα1-2[6OSO3]Galβ1-4GlcNAc-Sp0
267	Fucα1-2Galβ1-4[6OSO3]GlcNAc-Sp8
268	Fucα1-2[6OSO3]Galβ1-4[6OSO3]Glc-Sp0
269	Fucα1-2[6OSO3]Galβ1-4Glc-Sp0
270	Fucα1-2Galβ1-4[6OSO3]Glc-Sp0
271	Galβ1-3(Fucα1-4)GlcNAcβ1-3Galβ1-3(Fucα1-4)GlcNAcβ-Sp0
272	Galβ1-3(Galβ1-4GlcNacβ1-6)GalNAc-Sp14
273	Galβ1-3(GlcNacβ1-6)GalNAc-Sp14
274	Galβ1-3(Neu5Acα2-3Galβ1-4GlcNacβ1-6)GalNAcα-Sp14
275	Galβ1-3GalNAcα-Sp14
276	Galβ1-3GlcNAcβ1-3Galβ1-3GlcNAcβ-Sp0
277	Galβ1-4(Fucα1-3)[6OSO3]GlcNAc-Sp0
278	Galβ1-4(Fucα1-3)[6OSO3]Glc-Sp0
279	Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-3(Fucα1-4)GlcNAcβ-Sp0
280	Galβ1-4GlcNAcβ1-3Galβ1-3GlcNAcβ-Sp0
281	Neu5Acα2-3Galβ1-3GlcNAcβ1-3Galβ1-3GlcNAcβ-Sp0
282	Neu5Acα2-3Galβ1-4GlcNAcβ1-3Galβ1-3GlcNAcβ-Sp0
283	[3OSO3]Galβ1-4[6OSO3]GlcNAcβ-Sp0
284	[3OSO3][4OSO3]Galβ1-4GlcNAcβ-Sp0
285	[6OSO3]Galβ1-4[6OSO3]GlcNAcβ-Sp0
286	6-H2PO3Glcβ-Sp10
287	Galα1-3(Fucα1-2)Galβ-Sp18
288	Galα1-3GalNAcα-Sp16
289	Galβ1-3GalNAcα-Sp16
290	Galβ1-3(Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ1-6)GalNAcα-Sp14
291	Galβ1-3Galβ1-4GlcNAcβ-Sp8
292	Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
293	Galβ1-4GlcNAcβ1-3(Galβ1-4GlcNAcβ1-6)Galβ1-4GlcNAc-Sp0
294	Galβ1-4GlcNAcβ1-3(GlcNAcβ1-6)Galβ1-4GlcNAc-Sp0
295	Galβ1-4GlcNAcα1-6Galβ1-4GlcNAcβ-Sp0
296	Galβ1-4GlcNAcβ1-6Galβ1-4GlcNAcβ-Sp0
297	GalNAcα1-3(Fucα1-2)Galβ-Sp18
298	GalNAcα-Sp15
299	GalNAcβ1-3Galβ-Sp8
300	GlcAβ1-3GlcNAcβ-Sp8
301	GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
302	GlcNAcβ1-2Manα1-3(GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12

Downloaded from www.jbc.org by guest, on May 3, 2011

303	GlcNAcβ1-3Man-Sp10
304	GlcNAcβ1-4GlcNAcβ-Sp10
305	GlcNAcβ1-4GlcNAcβ-Sp12
306	HOOC(CH3)CH-3-O-GlcNAcβ1-4GlcNAcβ-Sp10
307	Manα1-3(Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
308	Manα1-6Manβ-Sp10
309	Manα1-6(Manα1-3)Manα1-6(Manα1-3)Manβ-Sp10
310	Manα1-2Manα1-2Manα1-3(Manα1-2Manα1-6(Manα1-3)Manα1-6)Manα-Sp9
311	Manα1-2Manα1-2Manα1-3(Manα1-2Manα1-6(Manα1-2Manα1-3)Manα1-6)Manα-Sp9
312	Neu5Acα2-3Galβ1-3(Neu5Acα2-3Galβ1-4GlcNAcβ1-6)GalNAcα-Sp14
313	Neu5Acα2-3Galβ1-3(Neu5Acα2-6)GalNAcα-Sp14
314	Neu5Acα2-3Galβ1-3GalNAcα-Sp14
315	Neu5Acα2-3Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
316	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
317	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
318	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-N(LT)AVL
319	Fucα1-2Galβ1-3GalNAcα-Sp14
320	Galβ1-3(Neu5Acα2-6)GalNAcα-Sp14
321	Galβ1-4GlcNAcβ1-3GalNAc-Sp14
322	Neu5Ac(9Ac)a2-3Galβ1-4GlcNAcβ-Sp0
323	Neu5Ac(9Ac)a2-3Galβ1-3GlcNAcβ-Sp0
324	Neu5Acα2-6Galβ1-4GlcNAcβ1-3Galβ1-3GlcNAcβ-Sp0
325	Neu5Acα2-3Galβ1-3(Fucα1-4)GlcNAcβ1-3Galβ1-3(Fucα1-4)GlcNAcβ-Sp0
326	Neu5Acα2-6Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
327	Galα1-4Galβ1-4GlcNAcβ1-3Galβ1-4Glcβ-Sp0
328	GalNAcβ1-3Galα1-4Galβ1-4GlcNAcβ1-3Galβ1-4Glcβ-Sp0
329	GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
330	GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
331	(Neu5Acα2-3-Galβ1-3)(((Neu5Acα2-3-Galβ1-4(Fucα1-3))GlcNAcβ1-6)GalNAc-Sp14
332	GlcNAcα1-4Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
333	GlcNAcα1-4Galβ1-4GlcNAcβ-Sp0
334	GlcNAcα1-4Galβ1-3GlcNAcβ-Sp0
335	GlcNAcα1-4Galβ1-4GlcNAcβ1-3Galβ1-4Glcβ-Sp0
336	GlcNAcα1-4Galβ1-4GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ-Sp0
337	GlcNAcα1-4Galβ1-4GlcNAcβ1-3Galβ1-4GlcNAcβ-Sp0
338	GlcNAcα1-4Galβ1-3GalNAc-Sp14
339	Manα1-3(Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
340	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3(Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
341	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-6Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
342	Neu5Acα2-6Galβ1-4GlcNAcβ1-2Manα1-3Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
343	Galβ1-4GlcNAcβ1-2Manα1-3Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
344	Galβ1-4GlcNAcβ1-2Manα1-6Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
345	Galβ1-4GlcNAcβ1-2Manα1-3(Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
346	GlcNAcβ1-2Manα1-3(GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4(Fucα1-6)GlcNAcβ-Sp22
347	Galβ1-4GlcNAcβ1-2Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4(Fucα1-6)GlcNAcβ-Sp22
348	Galβ1-3GlcNAcβ1-2Manα1-3(Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4(Fucα1-6)GlcNacβ-Sp22
349	Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-3(Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp19
350	[6OSO3]GlcNAcβ1-3Gal β1-4GlcNAc-β-Sp0

Downloaded from www.jbc.org by guest, on May 3, 2011

351	KDNa2-3Galβ1-4(Fucα1-3)GlcNAc-Sp0
352	KDNa2-6Galβ1-4GlcNAc-Sp0
353	KDNa2-3Galβ1-4Glc-Sp0
354	KDNa2-3Galβ1-3GalNAcα-Sp14
355	Fucα1-2Galβ1-3GlcNAcβ1-2Manα1-3(Fucα1-2Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
356	Fucα1-2Galβ1-4GlcNAcβ1-2Manα1-3(Fucα1-2Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
357	Fucα1-2Galβ1-4(Fucα1-3)GlcNAcβ1-2Manα1-3(Fucα1-2Galβ1-4(Fucα1-3)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAβ-Sp20
358	Galα1-3Galβ1-4GlcNAcβ1-2Manα1-3(Galα1-3Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
359	Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp12
360	Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-3(Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4(Fucα1-6)GlcNAcβ-Sp22
361	Neu5Acα2-6GlcNAcβ1-4GlcNAc-Sp21
362	Neu5Acα2-6GlcNAcβ1-4GlcNAcβ1-4GlcNAc-Sp21
363	Fucα1-2Galβ1-3GlcNAcβ1-3(Galβ1-4(Fucα1-3)GlcNAcβ1-6)Galβ1-4Glc-Sp21
364	Galβ1-4GlcNAcβ1-2(Galβ1-4GlcNAcβ1-4)Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp21
365	GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ1-2Manα1-3(GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
366	Galα1-3(Fucα1-2)Galβ1-4GlcNAcβ1-2Manα1-3(Galα1-3(Fucα1-2)Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
367	Galα1-3Galβ1-4(Fucα1-3)GlcNAcβ1-2Manα1-3(Galα1-3Galβ1-4(Fucα1-3)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
368	GalNAcα1-3(Fucα1-2)Galβ1-3GlcNAcβ1-2Manα1-3(GalNAcα1-3(Fucα1-2)Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
369	Galα1-3(Fucα1-2)Galβ1-3GlcNAcβ1-2Manα1-3(Galα1-3(Fucα1-2)Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp20
370	Fucα1-2Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-3(Fucα1-2Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAcβ-Sp19
371	NeuAcα2-3Galβ1-4GlcNAcβ1-3GalNAc-Sp14
372	NeuAcα2-6Galβ1-4GlcNAcβ1-3GalNAc-Sp14
373	Neu5Acα2-3Galβ1-4(Fucα1-3)GlcNAcβ1-3GalNAcα-Sp14
374	GalNAcβ1-4GlcNAcβ1-2Manα1-6(GalNAcβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
375	Galβ1-3GalNAcα1-3(Fucα1-2)Galβ1-4Glc-Sp14
376	Galβ1-3GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAc-Sp14
377	GlcNAcβ1-3GalNAcα-Sp14
378	GlcNAcβ1-6GalNAcα-Sp14
379	Galβ1-3GlcNacβ1-3(Galβ1-3GlcNacβ1-3Galβ1-4GlcNacβ1-6)Galβ1-4Glcβ-Sp0
380	Galβ1-3GlcNAcβ1-3(Galβ1-4(Fucα1-3)GlcNAcβ1-6)Galβ1-4Glc-Sp21
381	Fucα1-2Galβ1-3(Fucα1-4)GlcNAcβ1-3(Galβ1-4GlcNAcβ1-6)Galβ1-4Glc-Sp21
382	Fucα1-2Galβ1-3(Fucα1-4)GlcNAcβ1-3(Galβ1-4(Fucα1-3)GlcNAcβ1-6)Galβ1-4Glc-Sp21
383	Galβ1-3GlcNAcβ1-3(Galβ1-3GlcNAcβ1-3Galβ1-4(Fucα1-3)GlcNAcβ1-6)Galβ1-4Glc-Sp21
384	Galβ1-4GlcNacβ1-2(Galβ1-4GlcNacβ1-4)Manα1-3(Galβ1-4GlcNacβ1-2(Galβ1-4GlcNacβ1-6)Manα1-6)Manβ1-4GlcNacβ1-4GlcNacβ-Sp21
385	GlcNacβ1-2(GlcNacβ1-4)Manα1-3(GlcNacβ1-2Manα1-6)Manβ1-4GlcNacβ1-4GlcNac-Sp21
386	Fucα1-2Galβ1-3GalNAcα1-3(Fucα1-2)Galβ1-4Glcβ-Sp0
387	Fucα1-2Galβ1-3GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ-Sp0
388	Galβ1-3GlcNAcβ1-3GalNAcα-Sp14
389	Neu5Acα2-3(GalNAcβ1-4)Galβ1-4GlcNAcβ1-3GalNAcα-Sp14
390	GalNAcα1-3(Fucα1-2)Galβ1-3GalNAcα1-3(Fucα1-2)Galβ1-4GlcNAcβ-Sp0
391	Galα1-3Galβ1-3GlcNAcβ1-2Manα1-3(Galα1-3Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp19

Downloaded from www.jbc.org by guest, on May 3, 2011

392	Galα1-3Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-3(Galα1-3Galβ1-3(Fucα1-4)GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp19
393	Neu5Acα2-3Galβ1-3GlcNAcβ1-2Manα1-3(Neu5Acα2-3Galβ1-3GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp19
394	Galβ1-4GlcNAcβ1-2Manα1-3(GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
395	GlcNAcβ1-2Manα1-3(Galβ1-4GlcNAcβ1-2Manα1-6)Manβ1-4GlcNAcβ1-4GlcNAc-Sp12
396	Neu5Acα2-3Galβ1-3GlcNAcβ1-3GalNaca-Sp14
397	Fucα1-2Galβ1-4GlcNacβ1-3GalNaca-Sp14
398	Galβ1-4(Fucα1-3)GlcNacβ1-3GalNaca-Sp14
399	GalNaca1-3GalNacβ1-3Galα1-4Galβ1-4GlcNacβ-Sp0
400	Galα1-4Galβ1-3GlcNacβ1-2Manα1-3(Galα1-4Galβ1-3GlcNacβ1-2Manα1-6)Manβ1-4GlcNacβ1-4GlcNacβ-Sp19
401	Galα1-4Galβ1-4GlcNacβ1-2Manα1-3(Galα1-4Galβ1-4GlcNacβ1-2Manα1-6)Manβ1-4GlcNacβ1-4GlcNacβ-LVaNKT
402	Galα1-3Galβ1-4GlcNacβ1-3GalNaca-Sp14
403	Galβ1-3GlcNAcβ1-6Galβ1-4GlcNAcβ-Sp0
404	Galβ1-3GlcNAcα1-6Galβ1-4GlcNAcβ-Sp0
405	GalNAcβ1-3Galα1-6Galβ1-4Glcβ-Sp8
406	GlcNAcβ1-6(GlcNAcβ1-3)GalNAcα-Sp14

Downloaded from www.jbc.org by guest, on May 3, 2011