Isolation and Partial Characterization of Blood Group A and H Active Glycosphingolipids of Rat Small Intestine*  

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Blood group A and H active glycosphingolipids have been isolated from rat small intestine. By mass spectrometry of the permethylated and LiAlH₄-reduced permethylated glycolipid derivatives, the A glycolipids were shown to contain four (A-4), six (A-6), and 12 (A-12) sugar residues, respectively. The anomer structure of the A-4 and A-6 glycolipids was established by proton NMR spectroscopy of the permethylated-reduced derivatives. Acid degradation and gas chromatography were used for analysis of binding positions. The structures of the A-4 and A-6 glycolipids were GalNAcβ1 → 3Galβ1 → 4Glcβ1 → 1Cer and GalNAcα1 → 3Galβ1 → 4GlcNAcβ1 → 4Galα1 → 4Glcβ1 → 1Cer. The third glycolipid (A-12) was a branched dodecaglycosylceramide with two blood group A determinants. The complete structure of this glycolipid has not yet been solved. The blood group activity was the same for the A-6 and A-12 glycolipids based on an equal number of blood group A determinants, but the activity of the A-4 compound was only about 1% of the A-6 and A-12 compounds. The A-4 and A-6 glycolipids were also identified as the 12-sugar compound based on the mucosa epithelial cells. The blood group A activity of these two glycolipids was found to be identical. The three rat intestinal blood group A active glycolipids were exclusively located to the mucosa epithelial cells. The blood group H active tri- and pentaglycosylceramides (H-3 and H-5), presumed to be the precursors of the A-4 and A-6 glycolipids, were also identified. A 10-sugar glycolipid (H-10), a possible precursor of A-12, was not detected.

Glycosphingolipids with blood group A determinants have been isolated from both human and animal tissues. Five different glycolipid-based blood group A type carbohydrate determinants have so far been identified (1–9). Several of these are linked to glycosylceramide backbones of varying complexity as reviewed by McKibbin (7). The blood group A determinants found in human tissues are all built on type 1 (Galβ1 → 3GlcNAc) or type 2 (Galβ1 → 4GlcNAc) carbohydrate chains (7) but in animal tissues core saccharides lacking the group H active tri- and pentaglycosylceramides (H-3 and H-5) have also been identified (1, 2).

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1 Portions of this paper (including “Materials and Methods” and Figs. 2–8) are presented in miniprint as prepared by the authors. Miniprint is easily read with the aid of a standard magnifying glass. Full size photocopiables are available from the Journal of Biological Chemistry, 9650 Rockville Pike, Bethesda, MD 20814. Request Document No. 81M-1072, cite the authors, and include a check for $4.00 per set of photocopies. Full size photocopiables are also included in the microfilm edition of the Journal that is available from Waverly Press.
### RESULTS

The total non-acid glycolipid fraction isolated from black-white rat small intestine is shown in Fig. 1, lane T. All bands stained for carbohydrate (green) with the anisaldehyde reagent.

Several glycolipid bands were present having a thin layer mobility as for compounds from one up to 12 sugar residues. The total amount of non-acid glycolipids obtained from 120 animals was 1.2 g which corresponded to 6.8 mg/g of tissue (dry weight) or about 9 mg/animal.

#### Isolation of Pure Glycolipids—The A-4 glycolipid was purified by repeated column chromatography as acetylated derivative. The native glycolipid fraction, eluted with 0–15 volume % CH2 OH in CHCl3 from the silicic acid column described above, was acetylated. Thin layer chromatography revealed that the distance between the acetylated tri- and tetracyglycerolipids was widened compared to the native derivatives. This change in relative thin layer mobility was probably due to the amino sugar present in the tetracyglycerolipide fraction.

In between these two major compounds, a weak glycolipid band was detected. The acetylated mono- to tetracyglycerolipide fraction was applied on a LiChroprep column eluted by 0, 0.5, 1, 1.5, 2, and 75 volume % CH2 OH in CHCl3. The fraction eluted with 1.5 volume % contained A-4 and part of the ordinary tri- and tetracyglycerolipides. This component was further purified in a similar way on several LiChroprep columns until a pure single band was seen on the thin layer plate. After deacetylation, a major band was seen migrating as a tricyglycerolipide (Fig. 1, lane A-4). Two faint bands were also present migrating just in front of and behind the major compound. The weight of this fraction obtained from 80 animals was 4.6 mg. In addition, a 3.8-mg fraction was obtained which was contaminated by the tricyglycerolipide.

The A-6 glycolipid was isolated from 1.2 g of total non-acid glycolipids altogether (120 animals). The batch of glycolipids having five to eight carbohydrate residues, isolated with 15–30 volume % CH2 OH in CHCl3, by repeated silicic acid column chromatography as described above, was further fractionated on several silicic acid columns. Two fractions containing the six-sugar components were obtained partly pure. These were acetylated and the major components seen on thin layer chromatography were purified by repeated LiChroprep column chromatography. These glycolipids were eluted from the columns with 0–0.5 volume % CH2 OH in CHCl3 due to a higher ethanol contamination of the CHCl3 as noted above (compare the A-4 glycolipid). In this way, three acetylated glycolipid fractions were obtained which were pure or nearly pure as seen on the thin layer plate. After deacetylation, only one fraction was homogeneous (Fig. 1, lane A-6). The other two were contaminated with more slow moving components.

The weight of the pure glycolipid fraction was 11.3 mg but the total weight of the A-6 component in the total glycolipid fraction was estimated to about 40 mg.

The A-12 glycolipid was isolated from the native fraction eluted with 30–100 volume % CH2 OH in CHCl3 from the initial silicic acid column described above. This fraction was shown by thin layer chromatography to consist of a major component and several minor ones. The major glycolipid was purified by repeated silicic acid chromatography. The partly pure fraction was acetylated and further fractionated on LiChroprep columns. A pure fraction eluted with 1.75 to 2 volume % CH2 OH in CHCl3 was deacetylated, and this fraction was finally purified on a silicic acid column. 8.6 mg was obtained pure (Fig. 1, lane A-12) from 120 animals and, in addition, about 7 mg was present in nonpurer fractions.

During the preparation of the A-6 compound, a glycolipid fraction was eluted as acetylated derivative just in front of the A-6 glycolipid fraction. After deacetylation, this fraction showed a major band migrating as a five-sugar compound. It was further purified on a silicic acid column and a homogeneous fraction was eluted with 15 volume % CH2 OH in CHCl3. The weight of this fraction was 3.7 mg, isolated from 120 animals.

#### Structural Characterization of the A-4 Glycolipid—The mass spectra of the permethylated and permethylated-reduced A-4 glycolipids are shown in Figs. 2 and 3, respectively. A series of intense ions, formed by a loss of part of the long chain base, was found at m/e 1125 to 1237 (Fig. 3). These ions are evidence for four-sugar residues (1 Fuc, 1 hexosamine, and 2 hexoses) and non-hydroxy 16:0–24:0 fatty acids. A corresponding series of peaks was seen at m/e 1197 to 1309 for the nonreduced derivative (Fig. 2). Fragments due to the complete saccharide chain and part of the ceramide appeared at m/e 907...
915 (Fig. 2) and at m/e 901 (Fig. 3). Molecular ions for the species with trihydroxy 18:0 long chain base and non-hydroxy 16:0-24:0 fatty acid were present at m/e 1437 to 1549 and 1409 to 1521 (Figs. 2 and 3, respectively). Two types of terminal sugars were found. Terminal Fuc was seen at m/e 189 and 157 (189 minus methanol) in both spectra. The terminal hexosamine, seen at m/e 268 and 228 in the permethylated spectrum (Fig. 2), lost 14 mass units upon reduction and was present at m/e 246 and 262 in Fig. 3. The terminal trisaccharide was shown by m/e 638 in Fig. 2. Additional information about the carbohydrate sequence was given by the series of peaks at m/e 296 to 408, m/e 502 to 614, and m/e 880 to 992 in the spectrum of the reduced derivative (Fig. 3).

Ceramide fragments from the permethylated derivative (Fig. 2) were found at m/e 580 to 692 for 18:0 trihydroxy base in combination with 16:0-24:0 non-hydroxy fatty acid. Small amounts were present of 18:1 dihydroxy base and 24:0 non-hydroxy fatty acid (m/e 660) and 18:0 trihydroxy base in combination with 24:0 hydroxy fatty acid (m/e 722). The peak at m/e 396 was due to 18:0 trihydroxy base.

In addition to the A-4 glycolipid, small amounts of a contaminating trihexosylceramide were seen in the mass spectra. This was best evident in the reduced spectrum, where the peaks at m/e 924 (16:0 fatty acid), 980 (20:0), 1006 (22:0), and 1036 (24:0) were due to three hexoses and the fatty acid which is similar to the series at m/e 1125 to 1237 for the A-4 glycolipid. Terminal hexose was shown by m/e 219 and 187 (219 - 32). In the permethylated spectrum (Fig. 2), trihexosylceramide fragments were found at m/e 219, and 187 and at m/e 1038, 1066, and 1094. This contamination explained the weak band seen in front of the major A-4 band on the thin layer plate (Fig. 1, lane A-4). The major A-4 band consisted of the longer fatty acid (20:0 to 24:0) species and the weak band seen behind was due to the shorter fatty acid (16:0) species.

Degradation and gas chromatography of the native A-4 glycolipid showed the presence of aldito acetates of Fuc, Glc, Gal, and GalNAc with a molar ratio of about 1:1:1:1 (Table I) in agreement with the mass spectrometric data. The gas chromatogram of the degradation products obtained from the permethylated glycolipid fraction is shown in Fig. 4. chromatogram A. The peaks were identified as the acetates of 2.5:4-trimethylfucitol (designated Fuc1 →), 2,3,6-trimethylglucitol (→ 4Glc1 →), 4,6-dimethylgalactitol (→ 3Gal1 →), and 3,4,6-trimethyl-2-N-methylacetylaminodeoxyglactitol (GalNAc1 →). The peaks from the contaminating trihexosylceramide were very small, which made them impossible to identify among the by-product of degradation. The results of the degradation of the permethylated-reduced derivative are shown in Fig. 5, chromatogram A. The reduction of the amino sugar makes the glycosidic bond nearest the amino sugar nitrogen-resistant against degradation at the conditions used for the permethylated derivative, and therefore di- and trisaccharides are obtained (21, 22). Compared with the chromatogram of the permethylated derivative (Fig. 4A), the peaks due to the Gal (→ 2Gal1 →) and the GalNAc (GalNAc1 →) were lacking and, instead, a peak with a very long retention time had appeared (GalNAc1 → 3Gal2 →). The Fuc and Glc peaks were unchanged. This peak has been identified by mass spectrometry to be the acetate of 3-(3,4,6-trimethyl-2-N-methylacetylamido-2-deoxygalactopyranosyl)-4,6-dimethylgalactitol. The presence of this disaccharide excluded the possibility that Gal instead of Glc was located nearest the ceramide and also conclusively established that Fuc was bound to C-2 of the Gal and not to C-3.

The structural data presented so far established the type and sequence of sugars as well as the ceramide composition. Information on the anomery of the glycosidic bonds was given by proton NMR spectroscopy. The interpretation of NMR spectra of intact permethylated and permethylated-deproduced glycolipids was based on the fact that the coupling constants for α and β protons differ and that the chemical shifts of separate glycosidic bonds may differ (6, 23, 24). In addition, the reduction introduces a change of the chemical shift for some signals (24). The anomeric region of the NMR spectrum of the permethylated-reduced A-4 glycolipid is reproduced in Fig. 6, spectrum A. This spectrum showed a sharp signal of Fuc at 5.32 ppm (J₁,₂ = 3.4 Hz) due to an α proton. The signal seen at 5.09 ppm (J₁,₂ = 2.7 Hz) probably originated from H-1 of α-GalNAc (24). The doublet at 4.20 ppm (J₁,₂ = 7.4 Hz) was due to H-1 of β-Glc and the signal at 4.28 ppm came from H-1 of β-Gal. The peak at 5.15 ppm was due to a contamination of the CHCl₃ solvent.

The trihexosylceramide found as a contaminant by the mass spectra gave a signal at 4.94 ppm, due to H-1 of the terminal α-Gal (23). The two other signals of this glycolipid were buried in the β region at 4.28 and 4.20 ppm, respectively.

In conclusion, the structural studies of the A-4 glycolipid showed it to be a tetracyigosylceramide with a blood group A determinant and the following structure: GalNAcp1 → 3Galp(2 → 1aFucp)β1 → 4Glcβ1 → 1Cer. The ceramide part was made up of mainly 18:0 trihydroxy base and 16:0-24:0 non-hydroxy fatty acids.

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blood group A determinant and based on a type 1 carbohydrate chain. The complete structure was GalNACp\(\alpha\)1 → 3Galp\(\beta\)1 → 4Galp\(\beta\)1 → Cer. The ceramide part was made up of almost exclusively trihydroxy 18:0 long chain base in combination with both non-hydroxy and hydroxy 16:0–24:0 fatty acids.

This glycolipid inhibited the hemagglutination of human...
Blood Group A and H Active Glycolipids

A glycolipid fraction, having a thin layer mobility as a five-sugar compound, isolated during the preparation of the A-6 glycolipid (see above) was structurally characterized by mass spectrometry of the permethylated and permethylated-reduced derivatives, by NMR spectroscopy of the reduced derivative and by degradation—gas chromatography of the native and permethylated glycolipid. The structural data (not shown) established this fraction to be a mixture of two components. The major one was a blood group H type pentaglycosylceramide (FucGalα1-2Galβ1-3GalNAcβ1-4Glcβ1-1Cer), and the minor contained FucGalα1-2Galβ1-3Galβ1-4Glcβ1-1Cer. The ceramide was made up of trihydroxy 18:0 base and hydroxy 16:0−24:0 fatty acids. The probable structure of the minor glycolipid

**Table II**

| Antigen                          | Dilution of anti-A antiserum |
|----------------------------------|-----------------------------|
| Saline control                   | 1:1 1:2 1:4 1:8 1:16 1:32    |
| A-4 (3.9 µg/ml)                   | 4+ 4+ 4+ 3+ 2+ 1+            |
| A-6 (5.0 µg/ml)                   | 3+ 1+                         |
| A-12 (4.2 µg/ml)                  | 3+ 1+ (1+)                    |
| Human erythrocyte glycolipid (5.0 µg/ml) | 3+ 1+                        |

**Structural Characterization of the Blood Group H Type Precursors of the Blood Group A Glycolipids**—The blood group A glycolipids presented here are probably synthesized by enzymatic addition of the terminal GalNAc to the corresponding blood group H precursors (27, 28). In a recent paper, we described the presence of a blood group H active triglycosylceramide (H-3) isolated from the black-white rat small intestine (29). The structure was shown to be FucGalα1-2Galβ1-3Galβ1-4Glcβ1-1Cer.
component was Gal-GalNAc-Gal-Glc-Cer. The NMR spectrum indicated that this glycolipid may be gangliotetraosylceramide. It was not possible to separate these two species due to lack of material and a very close chromatographic pattern indicated that this glycolipid may be gangliotetraosylceramide. The third blood group A glycolipid was present in the large intestine of the black-white rat but has not been found in the small intestine (36).

Tissue Localization of Blood Group A and H Glycolipids—We have modified the technique of Welser (31) for isolation of mucous epithelial cells from rat small intestine (30, 32). The epithelial cells were completely removed from the intestinal stroma (32). The total non-acidic glycolipid fraction of the epithelial cells is shown in Fig. 1, lane C. Some glycolipids present in the total intestine were lacking in the epithelial cells. A detailed analysis of the epithelial cells and the residual stroma showed that blood group A and H glycolipids were exclusively present in the epithelial cells (30).

DISCUSSION

The glycolipid composition of rat small intestine is very complex and we have isolated about 30 different carbohydrate structures of which several were earlier unknown (30, 33). Three blood group A glycolipids with 4, 6, and 12 sugar residues and two blood group H compounds with 3 and 5 sugar residues were isolated from the small intestine of an inbred rat strain (black-white). In addition, a blood group H type compound with terminal Fuc linked to four hexoses was found in small amounts (33). The A-4 glycolipid was shown to have FucA→2 and GalNAc→3 linked to the Gal of lactosylceramide. The six-sugar compound (A-6) was based on a type 1 carbohydrate core structure which means FucA→2 and GalNAcA→3 linked to the terminal galactose of lactotetraosylceramide. The third blood group A glycolipid (A-12) was a branched dodecaglycosylceramide with two blood group A determinants.

The exact location of the terminal Fuc and GalNAc on the subterminal Gal of the A-4 and A-6 glycolipids was established by degradation and gas chromatography of the permethylated-reduced derivative. The reduction of the amide group of N-acetylglucosamine makes the glycosidic bond on C-1 of the amino sugar resistant against acid degradation at the experimental conditions used for permethylated glycolipids (21). The disaccharide obtained from the A-4 glycolipid showed that GalNAc was linked to C-3 of the subterminal Gal and therefore Fuc was located at the C-2 of the Gal. This information was also obtained for the A-6 compound by one of the two disaccharides obtained due to the presence of both GalNAc and GlcNAc. In addition, important information about the sequence of the sugars was given by these disaccharides.

The chemical identification of blood group A glycolipids was supported by their ability to inhibit hemagglutination of human red cells by anti-A antisera. The immunological activity was identical for the rat A-6 compound and the corresponding type 2 chain isomer of human erythrocyte. The reactivity of the A-6 and A-12 compounds was the same when corrected for the fact that each A-12 glycolipid contained two A saccharides. The A-4 glycolipid had weaker blood group A activity. The identical reactivity of type 1 and 2 chain A-6 compounds indicated that the GlcNAc is not specifically involved in the binding of the antibodies to the antigen. However, the addition of FucA1→4 by the Lewis enzyme to GlcNAc of the A-6 glycolipid or the corresponding blood group B-6 glycolipid completely abolishes the blood group activity (34) showing that some part of the antibody extends over the GlcNAc residue.

The A-4 glycolipid has not been described before and is so far the smallest blood group A active glycolipid isolated. Recently, a free tetrasaccharide was isolated from human urine and shown to have the same structure as that of the rat A-4 glycolipid (35). A corresponding blood group B active tetraglycosylceramide with terminal Gal instead of GalNAc was present in the large intestine of the black-white rat but has not been found in the small intestine (36).

The blood group A glycolipids present in the black-white rat small intestine are probably synthesized by addition of a terminal GalNAc to the corresponding blood group H precursors (7, 27, 28). Blood group H glycolipids having three (H-3) and five (H-5) sugars were present in the intestine but the branched 10-sugar compound (H-10) was lacking. The H-10 glycolipid was, however, present in another rat strain (white) together with the H-3 and H-5 glycolipids (30, 37). In this strain, blood group A glycolipids were completely lacking. This finding indicates that there may be different enzymes for the synthesis of the short and long carbohydrate chains or that the A-12 glycolipid is synthesized by a multiglycosyltransferase system which does not release the precursor. Other explanations may be that the short and long carbohydrate chain glycolipids are synthesized in different cell compartments or that the enzyme is more effective on longer carbohydrate chains and completely converts the blood group H determinant to A.

In relation to this, one may note that the ceramide composition of the A-4 and H-3 glycolipids differed. The A-4 glycolipid contained non-hydroxy fatty acids having a bimodal chain length distribution with predominantly 16:0 and 24:0 acids while the H-3 glycolipid had mainly non-hydroxy 20:3 and 22:0 acids (29). This may indicate that the H-3 is not the precursor for the A-4 glycolipid or that certain molecular species are selected by the glycosyltransferase.

In human erythrocyte blood group A type glycolipids with...
isolated epithelial cells and the residual intestinal stroma (30, 32). The biological significance of the cell, tissue, and individual specificity of the distribution of glycolipids remain to be shown.

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