Carbohydrate Structures of HVJ (Sendai Virus) Glycoproteins*

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The carbohydrate structures of two membrane glycoproteins (HANA protein and F protein) of HVJ have been determined on materials purified from virions grown in the allantoic sac of embryonated chicken eggs. Both glycoproteins contain fucose, mannose, galactose, and glucosamine but not galactosamine, indicating that their sugar chains are exclusively of the asparagine-linked type. The radioactive oligosaccharide fractions obtained from the two glycoproteins by hydrazinolysis followed by NaB\(^{3H}\) reduction gave quite distinct fractionation patterns after paper electrophoresis. More than 75% of the oligosaccharides from F protein were acidic and separated into at least four components by paper electrophoresis. Only 18% of the oligosaccharide from HANA protein was acidic single component. These acidic oligosaccharides could not be converted to neutral oligosaccharide by sialidase digestion.

Structural studies of the neutral oligosaccharide fractions from HANA and F proteins revealed that both of them are mixtures of a series of high mannose type oligosaccharides and of complex type oligosaccharides with Gal\(^{1}\)a\(^{4}\) (Fuc\(^{3}\)GlcNAc) group in their outer chain moieties.

It has been known that HVJ has an activity of inducing high frequency of cell to cell fusion. HVJ is a negative strand RNA virus with sizes ranging from 150-250 nm. Like other paramyxoviruses, it consists of an inner nucleocapsid surrounded by a membranous envelope covered with spikes of approximately 130-150 \(\mu\) in length. The envelope of HVJ virions grown in the allantoic sac of embryonated hen's eggs contain six structural proteins, which are readily separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis in the presence of thiol derivatives (1, 2). Three of them are glycoproteins. HANA protein with molecular size of 67,000 daltons has both hemagglutinating and sialidase activities and constructs HANA spikes of viral envelope (3). F1 protein (Mr = 51,000) and F2 protein (Mr = 15,000) show neither hemagglutinating nor sialidase activities. In the viral envelope, these glycoproteins occur as F protein which is composed of 1 mol each of F1 and F2 proteins connected by a disulfide bond. F protein constructs F spikes (3) and plays essential roles in hemolysis, cell fusion, and infectivity of virions (2-5).

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EXPERIMENTAL PROCEDURES

Isolation of the Typ Enveloping Glycoproteins of HVJ. HVJ were grown in the allantoic sac of embryonated hen's eggs and purified by NaB\(^{3H}\) reduction followed by electrophoresis on starch gel and silver staining. Portion of this paper (including "Experimental Procedures" and Figs. 1 and 5-8) are presented in miniprint as prepared by the authors. Miniprint is easily read with the aid of standard magnifying glass. Full size photocopies are available from the Journal of Biological Chemistry, 9650 Rockville Pike, Bethesda, MD 20014. Request Document No. 80M-2541, cite author(s), and include a check or money order for $5.60 per set of photocopies. Full size photocopies are also included in the microfilm edition of the Journal that is available from Waverly Press.

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preparied from Gal-GlcNAc-Man-GlcNAc-Fuc-GlcNAc by sequential digestion with β-galactosidase, β-xylosidase, α-L-fucosidase, and N-acetylgalactosaminidase, respectively. Fuc-N-acetylgalactosaminidase was isolated from urine of fumigato Galectin patients [15]. GlcNAc[1-4]Man[1-6]GlcNAc[1-6]Man[1-3]Man[1-4]GlcNAc[1-4]GlcNAc[1-6]Man-GlcNAc. GlcNAc[1-6]Man-GlcNAc[1-3]Man[1-6]GlcNAc[1-3]Man-GlcNAc and Man[1-6]GlcNAc[1-4]GlcNAc[1-6]Man-GlcNAc were prepared from Gal-GlcNAc, Man-GlcNAc, GlcNAc, and GlcNAc by sequential digestion with β-galactosidase, β-xylosidase, α-L-fucosidase, and N-acetylgalactosaminidase, respectively. Glucose oligomers were obtained by partial acid hydrolysis of the products as reported previously [15].

RESULTS

Fractionation of Oligosaccharides by Anionic Charges

When the radioactive oligosaccharide fractions obtained from HANA and F proteins are subjected to paper electrophoresis at pH 5.4, they gave quite distinct fractionation patterns (Fig. 2). More than 75% of the oligosaccharides from F protein were acidic, which could be separated into several components with different mobilities. In contrast, only 18% of the oligosaccharides from HANA protein were acidic components. Each peak in the electrophoretograms was eluted from paper with water as indicated by bars in Fig. 2. The yields of radioactive oligosaccharides FN, FA-1, FA-2, FA-3, FA-4, HN, and HA were 4.84, 7.52, 1.23, 2.83, 3.57, 5.71, and 1.29 x 10^6 cpm, respectively. Each component from the deuterium-labeled oligosaccharide fraction was also recovered from electrophoresis paper. An aliquot (2 x 10^4 cpm) of each radioactive oligosaccharide was hydrolyzed in 0.4 ml of 4 N HCl at 100 °C for 2 h, and the reaction mixture was freed from acid by repeated evaporation with water. The hydrolysate was N-acetylated and analyzed by paper electrophoresis using borate buffer as reported previously [24]. N-Acetylglucosaminol was the only radioactive component detected from all samples. Therefore, N-acetylglucosamine is located at the reducing termini of all oligosaccharides as expected from the hydrazinolyses of asparagine-linked sugar chains.

All five acidic oligosaccharides were completely resistant to sialidase digestion, indicating that the acidic nature of these oligosaccharides cannot be ascribed to sialic acid like most other asparagine-linked acidic sugar chains.

The abbreviations used are: GlcNAc, N-acetylglucosamine; XylNAc, N-acetylcyllosamine; subscript OT, NaBr3H1-reduced oligosaccharides; subscript OH, NaBr-reduced oligosaccharides. All sugars mentioned in this paper were of D-configuration except for fucose, which has an L-configuration.
Fig. 3. Bio-Gel P-4 column chromatography of neutral oligosaccharide fractions. The radioactivity in each tube (3.4 ml/tube) was determined by liquid scintillation spectrometer. Arrows, eluting positions of glucose oligomers; numbers, glucose units. HN fraction (A), FN fraction (B), in Fig. 2 are shown.

The yields of the six oligosaccharides (HN-I, -II, -III, -IV, -V1, and -V2) from FN fraction were 8.9, 2.31, 9.5 x 10^6, 5.0 x 10^6, 4.3 x 10^6, 5.8 x 10^6 cpm, respectively. The yields of the seven radioactive oligosaccharides (FN-I, -II, -III, -IV, -V1, -V2, and -V3) were 1.6 x 10^6, 2.3 x 10^6, 6.3 x 10^6, 1.8 x 10^6, 2.3 x 10^6, 1.20 x 10^6, and 6.1 x 10^6 cpm, respectively. Deuterium-labeled FN and HN fractions were also fractionated in the same manner.

Structural Studies of Oligosaccharides

High Mannose-type Sugar Chains—Among the 13 radioactive oligosaccharides, six oligosaccharides, FN-I, FN-II, HN-I, HN-II, HN-III, and HN-IV, were all converted to a radioactive trisaccharide with the same mobility in Bio-Gel P-4 column as authentic Manβ1→4GlcNAcβ1→4GlcNAc by jack bean α-mannosidase digestion (Fig. 5C). These trisaccharides were then converted to GlcNAcβ1→4GlcNAcβ1 and [3H]N-acetylgalcosaminol by sequential digestion with β-mannosidase and β-N-acetylhexosaminidase, respectively (Fig. 5, D and E). These results indicated that all these six oligosaccharides belong to high mannose type sugar chains: (Manα1→3),Manβ1→4GlcNAcβ1→GlcNAcβ1. From the size change of each oligosaccharide before (Fig. 5A) and after (Fig. 5C) jack bean α-mannosidase treatment, the n values of FN-I and HN-I were estimated as 4 and those of FN-II and HN-II as 5. In the same way, the n values of HN-III and HN-IV were estimated as 6 and 7, respectively. In the following analyses, FN-I and FN-II gave the same results as HN-I and HN-II, respectively. Therefore, the results of HN-I and HN-II will be documented to represent the four oligosaccharides. When incubated with Aspergillus a-mannosidase, HN-1 was not hydrolyzed at all (Fig. 5B). In contrast, HN-II, -III, and -IV were all converted to HN-I, releasing 1, 2, and 3 mannose residues, respectively (Fig. 5B). These results indicated that HN-I has no Manα1→2 residue in its outer chain moiety, while HN-II, -III, and -IV have 1, 2, and 3 Manα1→2 residues, respectively. Methylation analysis of HN-I (Table I) indicated that its structure should be either Manα1→6-Manα1→3-Manβ1→4GlcNAcβ1→4GlcNAcβ1 or Manα1→6-Manα1→3-Manβ1→4GlcNAcβ1→4GlcNAcβ1. After periodate oxidation (for detail, see "Experimental Procedures"), the radioactive HN-I was completely converted to Manα1→6-Manβ1→4GlcNAcβ1→4GlcNAcβ1→4GlcNAcβ1, which was identified by paper chromatography and methylation analysis and its degradation to GlcNAcβ1→4GlcNAcβ1 by the second periodate oxidation (data not shown). Therefore, the structure of HN-I can be written as shown in Fig. 9. That HN-I, -II, and -IV have Manα1→6-Manα1→3-Manα1→6-Manα1→3-Manβ1→4GlcNAcβ1→4GlcNAcβ1 as their core was also confirmed by the same analyses of the heptaitols (Fig. 5B) obtained by Aspergillus α-mannosidase digestion (data not shown).
Methylation analysis of the intact oligosaccharides indicated that HN-II, -III, and -IV have 1, 2, and 3 additional Manα1 → 2 residues, respectively, as estimated by Aspergillus α-mannosidase digestion. Therefore, the structures of HN-II, -III, and IV can be written as shown in Fig. 9.

Complex-type Sugar Chains—The mobilities of FN-III and -IV in Bio-Gel P-4 column were 11.3 and 12.1 glucose units, respectively (Fig. 6, A and H). The radioactive FN-IV was not hydrolyzed by β-galactosidase, β-N-acetylhexosaminidase, Bacillus α-fucosidase, or jack bean or Aspergillus α-mannosidases. However, α-fucosidase I from Almond emulsin released 1 fucosyl residue from FN-IV and converted it to a radioactive component with the same mobility as FN-III (data not shown). The radioactive component gave exactly the same results as FN-III in the following structural analyses.

When the radioactive FN-III was incubated with β-galactosidase, 1 galactose residue was released (Fig. 6C). Jack bean α-mannosidase digestion of the radioactive peak in Fig. 6C released 1 N-acetylglucosamine, and 1 fucose residue upon sequential digestion with β-mannosidase (Fig. 6E), β-N-acetylhexosaminidase (Fig. 6F), and Charonia lampas α-1-fucosidase (Fig. 6G), respectively. The final radioactive product in Fig. 6G was identified as N-acetylglucosaminyl by borate paper electrophoresis (data not shown). These results indicated that the structure of FN-III can be written as Galβ1 → 4GlcNAcβ1 → Manα1

| Table I | Molar ratio of additio acetates from hydrolysates of permethylated oligosaccharides HN-I, HN-II, HN-III, HN-IV, HN-V, and HN-V2 |
|---------|---------------------------------------------------------------|
| Methylated sugar | Oligosaccharide molar ratios |
| Fucitol | HN-I | HN-II | HN-III | HN-IV | HN-V | HN-V2 |
| 2,3,4-Tri-O-methyl(1,6-di-O-acetyl) | 0 | 0 | 0 | 0 | 0.8 | 1.8 |
| Galactitol | 2,3,4,6-Tetra-O-methyl(1,6-di-C-acetyl) | 0 | 0 | 0 | 0 | 1.8 | 1.9 |
| Mannitol | 2,3,4,6-Tetra-O-methyl(1,6-di-C-acetyl) | 3.2 | 3.0 | 3.2 | 3.1 | 0 | 0 |
| 4,6-Tri-O-methyl(1,6,2-tri-O-acetyl) | 0 | 1.1 | 2.3 | 2.8 | 2.2 | 2.1 |
| 2,4-Di-O-methyl(1,3,5,6-tetra-O-acetyl) | 2.0 | 2.0 | 2.0 | 2.0 | 1.0 | 1.0 |
| 2-N-Methylaceta-mido-2-deoxyglu- citol | 1,3,5-Tri-O-methyl(4,6-di-O-acetyl) | 0 | 0 | 0 | 0 | 0.6 | 0.6 |
| 1,3,5,6-Tetra-O-methyl(4-mon-O-acetyl) | 0.9 | 0.9 | 0.9 | 0.9 | 0.3 | 0.2 |
| 3,6-Di-O-methyl(1,4,5-tri-O-acetyl) | 0.9 | 0.8 | 0.9 | 0.9 | 2.7 | 1.9 |
| 6-Mono-O-methyl(1,3,4,5-tetra-O-acetyl) | 0 | 0 | 0 | 0 | 0.7 |

*Numbers were calculated by making the values of 2,4-di-O-methyl mannitol as 2.0 or 1.0.
TABLE II
Molar ratio of alditol acetates from hydrolysates of permethylated oligosaccharides FN-I, FN-II, FN-III + IV, FN-V1, FN-V2, FN-V3, and their enzyme digests

| Methylated sugar | Molar ratio of alditol acetates | Molar ratios* |
|------------------|-------------------------------|--------------|
|                  | FN-I  | FN-II | FN-III + IV | FN-V1 | FN-V2 | FN-V3 | Core portion (FN-V1)** | FN-V2, 3 (+Fuc) |
| Fucitol          | 0     | 0     | 1.3         | 0.9   | 1.6   | 2.7   | 0.8                      | 0.8               |
| Galactitol       | 0     | 0     | 0.9         | 1.9   | 2.1   | 1.9   | 0                        | 2.1               |
| Mannitol         | 2.9   | 2.7   | 1.2         | 0     | 0     | 0     | 2.2                      | 0                 |
| 2,4,6-Tri-O-methyl(1,2,5-tri-O-acetyl) | 0     | 1.2   | 1.1         | 1.9   | 2.0   | 1.8   | 0                        | 2.0               |
| 2,4-Di-O-methyl(1,3,5,6-tetra-O-acetyl) | 2.0   | 2.0   | 1.0         | 1.0   | 1.0   | 1.0   | 1.0                      | 1.0               |
| 2-N-Methylacetamido-2-deoxyglucitol | 0     | 0     | 0.6         | 0.8   | 0.7   | 0.8   | 0.9                      | 0.8               |
| 1,3,5-Tri-O-methyl(4,5-di-O-acetyl) | 0.8   | 0.9   | 0.2         | 0.2   | 0.2   | 0.1   | 0.1                      | 0.2               |
| 3,6-Di-O-methyl(1,4,5-tri-O-acetyl) | 0.9   | 0.9   | 1.3         | 2.7   | 1.9   | 1.1   | 0.8                      | 2.8               |
| 6-Mono-O-methyl(1,3,4,5-tetra-O-acetyl) | 0     | 0     | 0.6         | 0     | 0.7   | 1.6   | 0                        | 0                 |

* Numbers were calculated by making the values of 2,4-di-O-methyl mannitol as 2.0 or 1.0.

** Although only the data of the core portion of FN-V1 (a mixture of peaks in Fig. 7C) are listed, the core portions of FN-V2 and FN-V3 (a mixture of peaks in Fig. 8E) gave similar results.

GlcNAc in an approximate molar ratio of 4:1. The complete structures of these two oligosaccharides were determined as shown in Fig. 10 by methylation analysis. Methylation analysis of the mixture of two oligosaccharide core portions in Fig. 7C (Table II) indicated that they have structures Man₁ → 6(Mana₁ → 3)Manβ1 → 4GlcNAcβ1 → 4(Fucα1 → 6)GlcNAcαH and Man₁ → 6(Mana₁ → 3)Manβ1 → 4GlcNAcβ1 → 4GlcNAcαH. Methylation analysis of intact FN-V1 indicated that two Galβ1 → 4GlcNAc outer chains are linked at the C-2 position of 2-α-mannosyl residues of the core portions.

Both FN-V2 and FN-V3 were also separated into a major and a minor components (Fig. 8, A and B, respectively). They were not hydrolyzed by Bacillus α-fucosidase but were converted by almond emulsin α-fucosidase I digestion to two components with the same mobilities as those in FN-V1 (Fig. 8C). That the two components in Fig. 8C have the same structures as FN-V1 shown in Fig. 10 was confirmed by sequential exoglycosidase digestion (Fig. 8, D and E) and by methylation analysis as described above. Therefore, FN-V2 and FN-V3 should be monofucosyl and difucosyl derivative of FN-V1, respectively. Methylation analysis of intact FN-V3 (Table II) indicated that the 2 fucosyl residues are linked at the C-3 position of N-acetylglucosamine residues in the outer chain moieties (Fig. 10). This structure was also supported by the evidence that FN-V3 was completely resistant to β-galactosidase digestion (data not shown).

Methylation analysis of intact FN-V2 (Table II) indicated that it has 1 each of Galβ1 → 4(Fucα1 → 3)GlcNAc and Galβ1 → 4GlcNAc residues in its outer chain moiety. When the radioactive FN-V2 was incubated with a mixture of β-galactosidase and β-N-acetyhexosaminidase, the two components decreased their sizes by approximately 3 glucose units (Fig. 8F), indicating that a Galβ1 → 4GlcNAc residue was removed from both oligosaccharides. Approximately 40% of the exposed α-mannosyl residues in both oligosaccharides were removed by jack bean α-mannosidase digestion (Fig. 8G). Therefore,
40% of FN-V2 should have Galβ1 → 4(Fucα1 → 3)GlcNAc residue on the Manα1 → 6 side and 60% on the Manα1 → 3 side (Fig. 10).

Structural analyses on HN-V1 gave exactly the same results as FN-V1. The only difference was that the ratio of oligosaccharide with fucose to that without fucose was 3:2.

Structural analyses of HN-V2 gave the same results as FN-V2 except that the product corresponding to Fig. 8F was completely resistant to jack bean α-mannosidase digestion. Therefore, Galβ1 → 4(Fucα1 → 3)GlcNAc outer chain of the two components in HN-V2 should exclusively be linked to Manα1 → 3 side as shown in Fig. 9.

**DISCUSSION**

Analyses of the carbohydrate composition of HANA and F proteins confirmed the report of Kohama et al. (26) that both glycoproteins contain fucose, mannose, galactose, and glucosamine but no galactosamine and sialic acids (data not shown). Therefore, all sugar chains in these glycoproteins should exclusively occur as asparagine-linked sugar chains, which must be completely released as oligosaccharides by hydrazinolysis described in this paper. Structural study of the sugar chains

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**Fig. 9. Structures of the neutral oligosaccharides released from HANA protein.** The asterisks indicate the possible location of the Manα1 → 2 residues.

**Fig. 10. Structures of the neutral oligosaccharides released from F protein.** The asterisks indicate the possible location of the Manα1 → 2 residues.
of vesicular stomatitis virus glycoprotein grown in different lectin-resistant Chinese hamster ovary cell lines indicated that the sugar chains of the viral glycoprotein reflect those produced in the plasma membrane glycoproteins of host cells (27). However, the distinct difference in the oligosaccharide patterns found in the two different glycoproteins of HVJ indicates that the sugar chains of viral envelope glycoproteins are not always a simple replica of host membrane glycoproteins. A possible explanation for the detection of different oligosaccharide patterns in the two glycoproteins is that more than a single biosynthetic machinery of sugar chains exist in endoplasmic reticulum and in Golgi apparatus of host cells, and production of the two viral glycoproteins is segregated from each other until they reach the plasma membrane of host cells. Another possibility is that the processing of asparagine-linked sugar chains is affected by the structure of polypeptide moiety to which they are linked and consequently results in the formation of different oligosaccharide patterns on different protein molecules.

Since the acidic oligosaccharides liberated from HANA and F proteins were not converted to neutral oligosaccharides by sialidase digestion, their structures have not been elucidated yet. However, these oligosaccharides must also be linked to the polypeptide moieties of HANA and F proteins through GlcNAc → Asn linkage, because they have N-acetylglucosamine at their reducing ends. That the two viral glycoproteins contain anionic residues other than sialic acid in their sugar chains is interesting. Since the viral envelope is enriched with sialidase, the viral glycoproteins may have acquired in compensation another anionic residue resistant to sialidase digestion. Whatever the anionic residue may be, it might be specific to viral glycoproteins or may occur in very minor glycoproteins of host cells because sialic acids are the common acidic residues of most animal glycoproteins (28, 29).

The complex type sugar chains of both HANA and F proteins contain Galβ1 → 4(Fucα1 → 3)GlcNAc residues in their outer chain moieties. This trisaccharide residue is rarely found in animal glycoproteins. So far, three glycoproteins, human α1-acid glycoprotein (30), human parotid α-amylase (8), and human lactotransferrin (31), are shown to have asparagine-linked sugar chains with the particular trisaccharide residue. Consequently the functional role of these unusual outer chains is an interesting subject for future study. Comparative study of the distribution of the sugar chains shown in Fig. 10 in F, and F2 glycoproteins must also be performed before the functional role of sugar chains is considered.

It is well established that concanavalin A inhibits HVJ-induced cell fusion. From the structures of the sugar chains in HVJ glycoproteins elucidated in this paper, the mechanism of the effect of concanavalin A can be estimated. Since HANA protein contains mainly high mannose type sugar chains, which binds strongly with concanavalin A, the lectin may cover HANA protein inhibiting viral attachment to cell surface and succeeding cell fusion.

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REFERENCES

1. Shimizu, K., Hosaka, Y., and Shimizu, Y. K. (1972) J. Virol 9, 842-850.
2. Ishimia, M., and Ouchi, M. (1973) J. Virol. 12, 1457-1465.
3. Shimizu, K., Shimuzu, Y. K., Kohama, T., and Ishida, N. (1974) Virology 62, 90-101.
4. Schell, A., and Choppin, P. W. (1974) Virology 57, 475-490.
5. Hosaka, Y., Senba, T., and Fukui, K. (1974) J. Gen. Virol. 25, 391-404.
6. Okada, Y., and Murayama, F. (1966) Exp. Cell Res. 44, 527-551.
7. Mizuochoi, T., Yonemasu, K., Yamaishi, K., and Kobata, A. (1978) J. Biol. Chem. 253, 7404-7409.
8. Yamaishi, K., Tachibana, Y., Nakayama, T., Kitamura, M., Endo, Y., and Kobata, A. (1980) J. Biol. Chem. 255, 5635-5642.
9. Yoshima, H., Takasaki, S., and Kobata, A. (1980) J. Biol. Chem. 255, 10793-10804.
10. Nishigaki, M., Yamaishi, K., Matsuda, I., Arashima, S., and Kobata, A. (1978) J. Biochem. (Tokyo) 84, 823-834.
11. Yamaishi, K., Liang, C.-J., Funakoshi, S., and Kobata, A. (1981) J. Biol. Chem. 256, 1283-1289.
12. Hughes, R. C., and Jeamloz, R. W. (1964) Biochemistry 3, 1535-1543.
13. Paulson, J. C., Prieels, J.-P., Glasgow, L. R., and Hill, R. L. (1978) J. Biol. Chem. 253, 5617-5624.
14. Li, Y.-T., and Li, S.-C. (1972) Methods Enzymol. 28, 702-713.
15. Ichishima, E., Ari, M., and Kumagai, H. (1980) Setogaku 52, 919.
16. Yamaishi, K., Ichishima, E., Ari, M., and Kobata, A. (1980) Biochem. Biophys. Res. Commun. 96, 1335-1342.
17. Uchida, Y., Tsukada, Y., and Sugimori, T. (1974) Biochim. Biophys. Acta 350, 425-431.
18. Sugahara, K., Okumura, T., and Yamaishi, I. (1972) Biochim. Biophys. Acta 268, 486-496.
19. Nishigaki, M., Muramatsu, T., Kobata, A., and Maeyama, K. (1974) J. Biochem. (Tokyo) 75, 509-517.
20. Yoshima, H., Takasaki, S., Mega, S.-I., and Kobata, A. (1979) Arch. Biochem. Biophys. 194, 394-398.
21. Ogata, A.-M., Muramatsu, T., and Kobata, A. (1977) Arch. Biochem. Biophys. 181, 353-358.
22. Kochibe, N. (1973) J. Biochem. (Tokyo) 74, 1141-1149.
23. Mizuochi, T., Yamaishi, K., Fujikawa, K., Kishi, W., and Kobata, A. (1979) J. Biol. Chem. 254, 6419-6425.
24. Endo, Y., Yamaishi, K., Tachibana, Y., Tojo, S., and Kobata, A. (1979) J. Biochem. (Tokyo) 86, 659-673.
25. Takasaki, S., and Kobata, A. (1974) J. Biochem. (Tokyo) 76, 783-789.
26. Kohama, T., Shimizu, K., and Ishida, N. (1978) Virology 90, 226-234.
27. Hunt, L. A. (1980) J. Virol. 35, 362-370.
28. Kornfeld, R., and Kornfeld, S. (1976) Annu. Rev. Biochem. 45, 217-237.
29. Kobata, A. (1980) in 27th International Congress of Pure and Applied Chemistry (Valmavuori, A., ed) pp. 185-192, Pergamon Press, Oxford.
30. Fournet, B., Montereul, J., Strecker, G., Dorland, L., Haverkamp, J., Vliegenthart, J. F. G., Binette, J. P., and Schmidt, K. (1978) Biochemistry 17, 5206-5214.
31. Spik, G., Fournet, B., Cheron, A., Strecker, G., and Montereul, J. (1979) in Glycoconjugates (Schauer, R., Boer, P., Buddecke, E., Kramer, M. F., Vliegenthart, J. F. G. and Wiegandt, H. eds) pp. 21-22, Georg Thieme Publishers, Stuttgart.
32. Weber, K., and Osborn, M. (1969) J. Biol. Chem. 244, 4406-4412.