The Purification, Composition, and Specificity of the Anti-T Lectin from Peanut (Arachis hypogaea)*

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Peanut agglutinin was purified by affinity chromatography on Sepharose-c-aminocaproyl-β-D-galactopyranosylamine. The purified lectin obtained in a yield of 150 mg/100 g of defatted peanut was homogeneous on polyacrylamide gel electrophoresis, ultracentrifugation, and gel filtration. The intrinsic sedimentation coefficient (s\text{\textit{w}}\text{\textit{v}}) and the intrinsic diffusion coefficient (D\text{\textit{w}}\text{\textit{v}}) were estimated at pH 7.4 as 5.7 ± 0.1 S and 5.0 × 10^{-9} \text{cm}^2\text{ s}^{-1}, respectively. The molecular weight of the agglutinin, determined by sedimentation and diffusion and by gel filtration, was found to be 110,000. Disc gel electrophoresis and gel filtration, both in the presence of sodium dodecyl sulfate, gave a single component of $M_r = 27,500$ suggesting that the lectin is a tetramer composed of four subunits. Four alanine residues per 110,000 g were found by NH$_2$-terminal analysis and the sequence of the five NH$_2$-terminal amino acids was: Ala-Glu-Ser-Val-Thr. Each cycle in a sequenator gave a single amino acid, suggesting that the four subunits are identical.

Peanut agglutinin does not contain covalently bound sugar; it is devoid of cysteine and cystine, low in methionine, histidine, and tryptophan, but rich in acidic and hydroxyamino acids. The lectin agglutinated erythrocytes of human ABO blood types equally well, but only after they have been treated with neuraminidase. Of the monosaccharides tested for inhibition of hemagglutination only D-galactose and (β- and β-D-galactosides were active. High inhibitory activity was found with the disaccharide DGalβ(1→3)DGalNAc and with the desialylated glycoproteins: α1-acid glycoprotein, fetuin, glycophorin, and human blood group NN or MM antigen. These desialylated glycoproteins also reacted with the lectin to form precipitin bands in Ouchterlony double diffusion in agar.

Extracts of peanut (Arachis hypogaea) have been known for some time to agglutinate neuraminidase-treated human red blood cells (1, 2). The agglutinin was designated "anti-T agglutinin" since it gave the same immunological reaction as the anti-T antibody of mammalian sera which is responsible for T-polyagglutination occurring in several bacterial and viral infections (3, 4). In fact peanut extracts have been used for clinical determination of T-polyagglutinability (5), but no attempt has been made to obtain the lectin in pure, homogeneous form. Since the agglutinating activity of peanut extracts could be inhibited by galactose and by lactose (1, 2), we tried to purify the lectin by affinity chromatography on a column of Sepharose-c-aminocaproyl-β-galactopyranosylamine previously prepared by us for isolation of soybean agglutinin (6). Peanut agglutinin thus purified was obtained in a homogeneous form. In this paper we describe the procedure for the purification of peanut agglutinin as well as some of the physicochemical and biological properties of the purified lectin.

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EXPERIMENTAL PROCEDURE

Materials

Source of Lectin—Peanuts, variety Shulamit (1974 crop), were purchased from Hazera Co., Haifa, Israel.

Glycoproteins and Sugars—Lacto-N-tetraose, lacto-N-neotetraose, and T-antigen (prepared from isolated O,MM and O,NN antigens) were the gifts of Dr. G. F. Springer, Department of Immunology Research, Evanston Hospital, Northwestern University; α1-acid glycoprotein was a gift of Dr. K. Schmid, Boston University School of Medicine, and glycophorin of Dr. I. Kahane, Department of Clinical Microbiology, The Hebrew University Hadassah Medical School, Jerusalem. Fetuin was purchased from Gibco. Galβ(1→3)GalNAc was a gift of Dr. H. M. Flowers, Department of Biophysics, of this institute. N-Acetyllactosamine (Galβ(1→4)GlcNAc) was a gift from the late Dr. R. L. Katzman. All other sugars were of the highest quality commercially available.

Enzymes—Neuraminidase was obtained from Behringwerke AG, Marburg/Lahn, Germany, and trypsin from Worthington.

Chemicals—Sepharose 4B and Sephadex G-150 (superfine) were from Pharmacia, Uppsala, Sweden. Cyanogen bromide and all chemicals used for electrophoresis were from Eastman Chemicals. Sodium

1 The structures of the oligosaccharides mentioned in this paper are as follows: Lacto-N-tetraose: Galβ(1→3)GlcNAcβ(1→3)Galβ(1→4)Glc; lacto-N-neotetraose: Galβ(1→4)GlcNAcβ(1→3)Galβ(1→4)Glc. All sugars used in this study are in the pyranose form and of the α configuration unless otherwise noted.
dialyzed extensively, first against water and then against 0.9% NaCl. The precipitate was dissolved in water (150 ml) and precipitate which formed was allowed to settle overnight in the refrigerator. The precipitate was redissolved in 0.9% NaCl (500 ml) and the above procedure was repeated. Ammonium sulfate (285 g) was added to the combined supernatants (750 ml) to 60% saturation at 4°C, the precipitate which formed was allowed to settle overnight in the cold and then collected by centrifugation at 15,000 x g for 30 min. The precipitate was dissolved in water (150 ml) and dialyzed extensively, first against water and then against 0.9% NaCl. Insoluble material in the dialysis bag was removed by centrifugation (25,000 x g for 20 min) and the clear supernatant (225 ml) was applied at 4°C to a column (2.7 x 17 cm) of Sepharose-ε-aminoacyl β-galactopyranosylamine (6). The column, kept at 4°C, was eluted at a rate of 100 ml/hour, first with 0.9% NaCl (800 ml) until no significant amount of material absorbing at 280 nm was detected in the effluent (A_{280} < 0.05) and then with a solution of galactose (0.05 M in 0.9% NaCl). The fractions eluted with galactose and containing material absorbing at 280 nm were pooled, dialyzed extensively against water, centrifuged (25,000 x g, 20 min), and lyophilized. The elution pattern of the ammonium sulfate fraction on the affinity column is shown in Fig. 1. Usually 130 to 150 mg of purified peanut agglutinin with a specific hemagglutinating activity of 2,000 units/mg toward neuraminidase-treated human erythrocytes were obtained (for the definition of a unit of hemagglutinating activity, see Refs. 7 and 8). The recovery of the activity applied to the affinity column was higher than 80%, with an over-all yield of 87% of the activity present in the original extract and a 66-fold purification (Table I).

Homogeneity of Peanut Agglutinin—Disc electrophoresis at pH 8.9 (14), at pH 4.3 (15), and at pH 7.2 (16) (the latter in the presence of 0.1% (w/v) sodium dodecyl sulfate) of the purified agglutinin revealed a single protein band (Fig. 2). Gel filtration on Sephadex G-150 (superfine) gave a single symmetrical protein peak with constant specific activity throughout (2,000 hemagglutinating units/mg). In sedimentation velocity studies in the ultracentrifuge, peanut agglutinin (14 mg/ml in buffer centrifuged at 56,000 rpm, 20°C) afforded a single symmetrical peak.

Molecular Weight—Gel filtration of peanut agglutinin on a column of Sephadex G-150 (superfine) which had been calibrated with proteins of known molecular weight, gave a value of 110,000 ± 10,000 relative to bovine γ-globulin (M_r = 150,000), soybean agglutinin (M_r = 120,000), bovine serum albumin (M_r = 68,000), ovalbumin (M_r = 45,000), and chymotrypsinogen (M_r = 25,000) (Fig. 3).

Sedimentation velocity studies gave a similar value, 111,000, which was calculated from the intrinsic sedimentation coefficient S_{20, w} = 5.7 ± 0.1 S and the intrinsic sedimentation coefficient D_{20, w} = 5.0 x 10^{-7} cm^2 s^{-1} assuming a partial specific volume of ϱ = 0.73 ml g^{-1} (calculated (17) from the amino acid composition given below).

Dissociation into Subunits—In disc gel electrophoresis, in the presence of 0.1% sodium dodecyl sulfate (16), peanut agglutinin migrated to a distance corresponding to a molecular weight of 27,000 ± 1,500 relative to bovine serum albumin (M_r = 68,000), ovalbumin (M_r = 46,000), glyceraldehyde phosphate dehydrogenase (M_r = 36,000), soybean agglutinin (M_r = 30,000), chymotrypsinogen (M_r = 25,000), and lysozyme (M_r = 14,400) (Fig. 4). Gel filtration on Sephadex G-150 (superfine) in the presence of 0.1% sodium dodecyl sulfate afforded a single peak of protein which emerged from the column at a position corresponding to a molecular weight of 28,000 ± 2,500 relative to catalase (M_r = 66,000), ovalbumin (M_r = 46,000), soybean agglutinin (M_r = 30,000), chymotrypsinogen (M_r = 25,000), and cytochrome c (M_r = 12,400) (Fig. 3).

These results indicate that peanut agglutinin is a tetramer composed of four apparently identical subunits.

Amino Acid Composition and Carbohydrate Analysis—Amino acid analysis of the agglutinin (Table II) revealed a particularly high content of acidic and hydroxyamino acids, a low content of methionine, tryptophan, and histidine, and the...
Affinity chromatography of peanut agglutinin on a column of Sepharose-γ-aminocaproyl-β-D-galactopyranosylamine. The ammonium sulfate (60%) fraction (225 ml) obtained from an extract of 100 g of defatted peanut was applied to a column (2.7 x 17 cm) and after washing out the unbound proteins with 800 ml of 0.9% NaCl (20-ml fractions), the lectin was eluted (10-ml fractions) with 0.05 M β-D-galactose in 0.9% NaCl. The arrow denotes the point of application of the galactose solution. The flow rate was 100 ml/hour and chromatography was carried out at 4°. For the determination of hemagglutinating activity (H.A.) samples were dialyzed extensively against 0.9% NaCl.

FIG. 2 (right). Disc electrophoresis of agglutinin on polyacrylamide gels. The direction of migration was from the top. Electrophoresis was performed in 7.5% acrylamide gels with 100 pg of peanut agglutinin at pH 8.9 at 1 ma for 5 hours (a); at pH 4.3 at 3 ma for 4 hours (b); and in the presence of 0.1% sodium dodecyl sulfate at 8 ma for 4 hours (c). The gels were stained for protein with Coomassie brilliant blue R-250.

TABLE I

<table>
<thead>
<tr>
<th>Purification of peanut agglutinin</th>
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<tbody>
<tr>
<td>Fraction*</td>
</tr>
<tr>
<td>Crude extract from 100 g defatted peanut</td>
</tr>
<tr>
<td>Ammonium sulfate (60%) precipitate</td>
</tr>
<tr>
<td>Galactose eluate from affinity column</td>
</tr>
</tbody>
</table>

*Each fraction was dialyzed extensively against 0.9% NaCl before determination of activity.

Protein in the first two fractions was determined by the method of Lowry et al. (10) and in the last fraction by weighing the lectin after dialysis against water and lyophilization.

NH₂-terminal Sequence—Quantitative determination of NH₂-terminal amino acids gave only dinitrophenylalanine in an amount corresponding to 0.8 mol/27,500 g of peanut agglutinin. The sequence of the five NH₂-terminal amino acids was found to be: alanine, glutamic acid, serine, valine, and threonine. Each cycle in the sequenator gave a single amino acid suggesting that the four subunits of the lectin are identical.

Hemagglutinating Activity—The lectin did not agglutinate untreated or trypsin-treated human erythrocytes whether type A, B, or O. However, after treatment of the erythrocyte with neuraminidase all the above erythrocytes were highly susceptible to agglutination by peanut agglutinin (2,000 ± 300 HU/mg).

**Inhibition Studies**—The affinity of peanut agglutinin for various mono-, di-, and oligosaccharides as well as for the circulating glycoproteins fetuin and α₁-acid glycoprotein and the human erythrocyte membrane glycoproteins, glycophorin, and T-antigen, was probed by measuring their ability to inhibit hemagglutination of human type B, neuraminidase-treated human erythrocytes. When plotted as percentage of inhibition versus the inhibitor concentration on a logarithmic scale (data not shown), the inhibition curves showed a characteristic

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*The abbreviation used is: HU, hemagglutinating units.
The disaccharide Galf(1→3)GalNac was a much better inhibitor of hemagglutination than any of the above saccharides (Table IIIC). The most potent inhibitors among these glycoproteins were desialylated lacto-1→4-lacto-N-neotetraose and its α- and β-galactosides (1.66-fold) than galactose. All the tetrasaccharides, lacto-1→4-tetraose and lacto-1→4-neotetraose were slightly more potent inhibitors (30-fold) than the above di- and tetrasaccharides. All the tetrasaccharides, lacto-1→4-tetraose and lacto-1→4-neotetraose were slightly more potent inhibitors than galactose (27.3) while lactose, N-acetyllactosamine, and the disaccharide Gal(1→3)GalNac were also inhibitory. Of the di- and oligosaccharides tested (Table IIIB), melibiose was the poorest inhibitor (less than galactose) while lactose, N-acetyllactosamine, and the tetrasaccharides, lacto-N-tetraose and lacto-N-neotetraose were slightly more potent inhibitors (1.66-fold) than galactose. The disaccharide Gal(1→3)GalNac was a much better inhibitor of hemagglutination than any of the above saccharides (Table IIIC). The most potent inhibitors among these glycoproteins were desialylated glycoprotein and the T-antigen.

The following monosaccharides did not exhibit any inhibitory activity at 0.25 M concentration: galactose 6-sulfate; 2-deoxygalactose, galactitol, γ-galactosylactone, calcium galactonate, UDP-galactose, galacturonic acid, N-acetylgalactosamine, β-nitrophenyl α- or β-N-acetylgalactosamine, glucose, methyl-α-glucoside, glucosamine, N-acetylgalactosamine, β-nitrophenyl α- or β-N-acetylgalactosamine, mannose, methyl-α-mannoside, N-acetylmannosamine, L-arabinose, xylose, D- or L-fucose, and rhamnose. Native (not desialylated) α- and β-galactoside, glycoprotein, fetuin, and glycophorin were not inhibitory.

**DISCUSSION**

This report is the first to describe the isolation and characterization of the lectin from peanut (Arachis hypogaea). For the purification of peanut agglutinin we used the affinity adsorbent Sepharose-ε-aminocaproyl-α-galactopyranosylamine (6), which is routinely employed in our laboratory for the purification of soybean agglutinin, another galactose-specific lectin. The purification procedure described herein afforded a lectin which was homogeneous by a variety of techniques. Peanut agglutinin is similar in its molecular weight (110,000) and subunit structure (four subunits) to a number of other purified lectins such as soybean agglutinin and wax bean agglutinin (26). Furthermore, the abundance of acidic and

**FIG. 3.** Determination of the molecular weight of peanut agglutinin (PNA) by gel filtration. A column (1.9 x 50 cm) of Sephadex G-150 (superfine) was used with 0.9% NaCl as eluent at 4°C (O--O), or with 0.1% sodium dodecyl sulfate in 0.9% NaCl at 23°C (●●●). The protein solution (1 ml, 3 to 5 mg/ml in either 0.9% NaCl or in 0.1% sodium dodecyl sulfate in 0.9% NaCl) was applied to the column and eluted at a rate of 8 ml/hour. Two-milliliter fractions were collected and proteins were located spectrophotometrically (at 230 nm or at 280 nm). Elution volumes were determined from the position of the maxima of the elution profiles.

**FIG. 4.** Determination of the subunit molecular weight of peanut agglutinin (PNA) by electrophoresis in polyacrylamide gel. Standard protein samples (50 μg each) were applied to the gel and after staining with Coomassie brilliant blue R-250 their mobility was measured from the top of the gel to the leading edge of the protein band.
TABLE III
Inhibitory effect of various sugars and glycoproteins on hemagglutinating activity of peanut agglutinin

<table>
<thead>
<tr>
<th>Sugar</th>
<th>Concentration needed for 50% inhibition (mM)</th>
<th>Relative inhibitory activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Monosaccharides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Galactose</td>
<td>100</td>
<td>1</td>
</tr>
<tr>
<td>Methyl-α-galactopyranoside</td>
<td>40</td>
<td>2.5</td>
</tr>
<tr>
<td>Methyl-β-galactopyranoside</td>
<td>90</td>
<td>1.25</td>
</tr>
<tr>
<td>α-Nitrophenyl-α-galactopyranoside</td>
<td>80</td>
<td>1.85</td>
</tr>
<tr>
<td>β-Nitrophenyl-β-galactopyranoside</td>
<td>80</td>
<td>1.25</td>
</tr>
<tr>
<td>Galactosamine</td>
<td>70</td>
<td>1.43</td>
</tr>
<tr>
<td>B. Di- and tetrascarbohydrates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Melibiose</td>
<td>125</td>
<td>0.8</td>
</tr>
<tr>
<td>Lactose</td>
<td>60</td>
<td>1.66</td>
</tr>
<tr>
<td>N-Acetyllactosamine</td>
<td>60</td>
<td>1.66</td>
</tr>
<tr>
<td>Lacto-N-tetraoseβ</td>
<td>60</td>
<td>1.66</td>
</tr>
<tr>
<td>Lacto-N-neotetraoseβ</td>
<td>60</td>
<td>1.66</td>
</tr>
<tr>
<td>Galβ(1→3)GalNAc</td>
<td>2</td>
<td>50</td>
</tr>
<tr>
<td>C. Glycoproteins (desialylated)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>α1-Acid glycoprotein</td>
<td>0.4</td>
<td>20</td>
</tr>
<tr>
<td>Fetuin</td>
<td>1</td>
<td>1,000</td>
</tr>
<tr>
<td>Glycophorin</td>
<td>0.01</td>
<td>10,000</td>
</tr>
<tr>
<td>T-Antigen (desialylated O,NN blood group substance)</td>
<td>0.008</td>
<td>12,500</td>
</tr>
</tbody>
</table>

*The concentration of inhibitors before the addition of 1 ml of neuraminidase-treated human type B erythrocyte suspension.

**The hemagglutination inhibitory activity of galactose was taken as 1.

† Due to scarcity of material the inhibition was assayed in microtiter plates (21) and the results were calculated in relation to the inhibitory activity of galactose in the same assay system.

‡ For comparison, the concentration of inhibitors has been normalized to their molar galactose content as given in the literature for α1-acid glycoprotein (22), fetuin (23), glycophorin (24), and NN blood group substance (25).

hydroxylamino acids and the paucity of sulfur-containing amino acids found in peanut agglutinin have also been reported for other lectins (26). However, unlike most of the lectins which are glycoproteins, peanut agglutinin does not contain any covalently bound carbohydrate.

Our inhibition experiments (Table III) confirm the original observations of Bird (1) and Uhlenbruck et al. (2), made with crude extracts of peanut, that galactose and lactose are poor inhibitors of peanut agglutinin and that the disaccharide Galβ(1→3)GalNAc and the desialylated human erythrocyte N-substance (T-antigen) are very potent inhibitors of this lectin.

We have extended the study to many other saccharides and in contrast to the suggestion of Uhlenbruck et al. (2) that only β-galactosides are inhibitors, we find that both α- and β-galactosides possess inhibitory activity.

Assuming that the hemagglutination inhibition activity of saccharides reflects the affinity of peanut agglutinin for the saccharides, it is possible to specify the structural requirements of the lectin combining site. Among galactosides differing only in the configuration about anomeric C-1, there seems to be a slight preference for the α anomers, except for melibiose which is less active than lactose or galactose. The aglycon of the galactoside (α- or β-D-nitrophenoxy and α- or β-methyl groups) does not influence markedly binding of the sugar by peanut agglutinin. Furthermore, aromatic aglycons of galacto-

sides do not increase the binding of the corresponding sugars by the lectin, which excludes the existence of a hydrophobic region adjacent to the monosaccharide binding site. Such a site was proposed for concanavalin A (27) and for soybean agglutinin (28). While galactosamine was a somewhat better inhibitor than galactose, N-acetylglactosamine and 2-deoxygalactose were not inhibitors, indicating a requirement for a free hydroxyl or a free amino group at C-2. In this respect the specificity of peanut agglutinin differs from soybean agglutinin which is better inhibited by N-acetylglactosamine than by galactose (29). The configuration at C-4 is also important for binding, since neither glucose nor methyl-glucoside were inhibitors. A C-6 hydroxymethyl group is essential for binding as galactose 6-sulfate, galacturonic acid, fucose, and L-arabinose, which are all isomeric with D-galactose at positions C-2, C-3, and C-4, but lack a free hydroxyl group at C-6, were not inhibitors.

Peanut agglutinin binds sugars in the pyranose form, since methyl α-galactopyranoside which is fixed in the six-membered ring form is a better inhibitor than galactose which exists as an equilibrium mixture of pyranose, furanose, and the open chain form. Galactose in turn is a better inhibitor than the straight chain galactitol which is not inhibitory at all.

The hypothetical structure of the sugar binding site of peanut agglutinin is demonstrated schematically in Fig. 6.

The finding that the disaccharide Galβ(1→3)GalNAc is a much better inhibitor than both galactose and Galβ(1→4)GlcNAc suggests that the agglutinin recognizes both the terminal nonreducing galactose residue as well as the penultimate GalNAc residue (which is not inhibitory). This trend of recognition is also reflected in the inhibitory activity of the desialylated glycoproteins tested: α1-acid glycoprotein contains Gal-GlcNAc, but not Gal-GalNAc sequences (22), and indeed it is a less potent inhibitor than fetuin which has been recently shown to contain Galβ(1→3)GalNAc sequences (23). The latter is less potent an inhibitor than the T-antigen which has only Gal-GalNAc sequences (30). Glycophorin carries the MN determinants (24) which after neuraminidase treatment are transformed into T-antigen with a Gal-GalNAc sequence and indeed desialylated glycophorin is a very potent inhibitor of peanut agglutinin.

The increased affinity of peanut agglutinin for Gal-GalNAc as compared with galactose may indicate the presence of an
extended combining site (Fig. 6). The presence of extended sugar binding sites has also been proposed for concanavalin A (31) for wheat germ agglutinin (32), and for the lectin from potato (33).

Peanut agglutinin unlike most of the lectins described thus far does not agglutinate untreated or trypsin-treated human erythrocytes. Preliminary experiments indicate that untreated cells bind very little[^1]-labeled agglutinin; however, after treatment of the cells with neuraminidase there is a great enhancement in the binding of the lectin and the erythrocytes are agglutinated. Similar results were obtained for the binding of peanut agglutinin to lymphocytes from a variety of sources and it was observed that the lectin was mitogenic to human peripheral blood lymphocytes and to rat lymphocytes only after the cells have been treated with neuraminidase.[^2]

The availability of a newly purified galactose-specific lectin with a sugar specificity somewhat different from that of soybean agglutinin makes it possible to gain further insight into the role of galactosyl residues on lymphocyte surface membrane in triggering blastogenesis. The purified peanut agglutinin may be used for the clinical determination of T-polyagglutinability of erythrocytes (5) and for probing cell membranes of normal and malignant cells. Recently Springer et al. (34) have reported that membranes from cells of malignant human breast glands absorbed the agglutinating principle from peanut extracts more efficiently than membranes from cells of healthy mammary glands and benign lesions. The purified peanut agglutinin may thus be of great use in the characterization of the tumor-specific antigens on the surface of malignant cells.


[^2]: The sites of protein-sugar interactions are marked. The presence of extended galactosyl residues on lymphocyte surface membrane in triggering blastogenesis. The purified peanut agglutinin makes it possible to gain further insight into the role of galactosyl residues on lymphocyte surface membrane in triggering blastogenesis. The purified peanut agglutinin may be used for the clinical determination of T-polyagglutinability of erythrocytes (5) and for probing cell membranes of normal and malignant cells. Recently Springer et al. (34) have reported that membranes from cells of malignant human breast glands absorbed the agglutinating principle from peanut extracts more efficiently than membranes from cells of healthy mammary glands and benign lesions. The purified peanut agglutinin may thus be of great use in the characterization of the tumor-specific antigens on the surface of malignant cells.

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