Monosaccharide Transport in Protein-depleted Vesicles from Erythrocyte Membranes*

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Treatment of human erythrocyte membranes with dilute alkali (pH 11.5) generates sealed, protein-depleted vesicles that can be isolated by density gradient centrifugation. The vesicles are 0.5 to 2.0 µm in diameter, and their membranes are predominantly oriented inside-out. The vesicles lack protein bands 1, 2, 5, and 6 (nomenclature of Steck, T. L. (1974) J. Cell Biol. 62, 1-19) of the erythrocyte membrane. L-Sorbose, a substrate of the monosaccharide transport system in erythrocytes, is transported by the vesicles. Based on comparisons between erythrocytes and vesicles with regard to specificity, temperature dependence, and effects of inhibitors, we conclude that sorbose uptake into the vesicles occurs by way of the monosaccharide transport system. The specific activity of the transport system in vesicles, as determined by initial rate measurements of sorbose uptake, averaged 58% of that in erythrocytes. This finding indicates that the major polypeptides of Bands 1, 2, 5, and 6 do not play an obligatory role in monosaccharide transport.

The monosaccharide transport system of the human erythrocyte catalyzes the equilibration of D-glucose and other sugars across the cellular membrane. A great deal is known about the kinetics, specificity, and inhibition of this system of facilitated diffusion (1-3). However, neither the identity of the erythrocyte membrane protein that is the major component of this system nor the possible role(s) of other erythrocyte membrane proteins in modulating the activity of the system has been, as yet, unequivocally determined (2, 3). As an approach to this problem, we have made use of the finding of Steck and Yu that treatment of erythrocyte membranes (ghosts) with dilute alkali selectively elutes four of the major proteins (Bands 1, 2, 5, and 6) (4). We have isolated sealed vesicles from the membrane pellet after alkali treatment and compared the activity of the monosaccharide transport system in these vesicles with that in intact erythrocytes.

EXPERIMENTAL PROCEDURES

Materials - Freshly outdated units of blood in citrate/dextran or citrate/pHosphate/dextran were kindly provided by the blood bank of the Mary Hitchcock Memorial Hospital, Hanover, N. H. Phloretin, obtained from Gallard-Schlesinger, was twice recrystallized from dilute ethanol. The following compounds were used without further purification: cytochalasin B (Aldrich), L-$^4$Hsorbose (Amersham/Searle), myo-$^2$Hinositol (New England Nuclear), dextran T-70 (Pharmacia).

Preparation of Protein-depleted Vesicles - Erythrocyte ghosts in 5 mm sodium phosphate, pH 7, were prepared from blood by the method of Steck and Kant (5). Packed ghosts (40 ml) (about 110 mg of protein) were added with stirring to 280 ml of water, and the pH was raised to 11.5-11.6 by the addition of about 0.4 ml of 1 N NaOH. This operation was performed with a pH meter that had been carefully standardized at pH values of 9.39 and 12.00 through the use of 10 mm K$_2$B$_4$O$_7$.5H$_2$O and 25 mm sodium phosphate buffers, respectively, at 5°. Immediately after adjustment of the pH, the suspension was centrifuged at 40,000 x g for 20 min. The supernatant was discarded and the pellet was resuspended in 40 ml of 2.6 mm sodium phosphate, pH 8.0. The pH was readjusted to 8.0 with 2.6 mm disodium phosphate and the membrane fragments were separated again by centrifugation. Vesicles were isolated from the pellet by density gradient centrifugation after the method of Steck for the isolation of sealed erythrocyte membrane vesicles (7). The pellet was resuspended to give 9 ml in 2.6 mm sodium phosphate, pH 8, and 1.5-ml aliquots were layered on top of 11.3 ml of a 1.00 to 1.06 g/cm$^3$ linear gradient of dextran T-70 in 0.5 mM sodium phosphate, pH 8.0. Centrifugation was carried out for 90 min at 37,000 rpm in a Beckman SW41 Ti rotor. The protein-depleted vesicles were collected from the gradient and washed two times by suspension in 40 ml of 2.6 mm sodium phosphate, pH 8, followed by centrifugation at 48,000 x g for 15 min. All the above operations were carried out at 0-3°. The vesicles were finally adjusted to a protein concentration of 1.4 mg/ml in 2.6 mM sodium phosphate, pH 8, and stored at 3°. SDS-polyacrylamide gel electrophoresis of the vesicles, after storage for various periods at 12.5°, revealed the disappearance of less than 10% of Band 3 after 24 h, of about 20% after 53 h, and of 60% after 78 h, probably as the result of the action of a protease in the preparation (8). The transport activity of the vesicles (see below) did not change over the initial 20 h after preparation, and the vesicles were routinely used for transport experiments within 17 h.

Rates of Transport into Vesicles - The usual procedure for measuring the uptake of L-sorbose by vesicles was the following. Vesicles (55.5 mg of protein) in 0.39 ml of 2.6 mM sodium phosphate, pH 8, were temperature-equilibrated in a water bath for 15 min. Ten microliters of 20 mM L-$^4$Hsorbose (1 µCi) was added with thorough mixing. At timed intervals thereafter, 50-µl aliquots were removed and quenched in 1 ml of 0.5 mM L-sorbose/2.6 mM sodium phosphate, pH 8, at 2°. Each quenched sample was immediately filtered through a 0.45-µm Millipore filter (HAWP 02500), and the filter was washed three times with 2 ml of quench solution in about 45 s. The radioactivity associated with the filters was determined by shaking the filters overnight with 10 ml of scintillation fluid (50 ml of Beckman BBS 3 and 4 gm of 2,5 diphenyloxazole (PPO) in 1 liter of toluene) and counting in a Packard Tri-Carb liquid scintillation spectrometer.

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1 The abbreviation used is: SDS, sodium dodecyl sulfate.
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A number of control experiments were performed in order to determine that this method gave true values for the uptake of L-sorbose. If the vesicles were allowed to remain in the quench medium for 1 min before filtration, there was no decrease in the amount of sorbose associated with the filter. Consequently, a significant fraction of the intravesicular sorbose does not exit during the filtration procedure. (b) Analysis of the filters by the Lowry method revealed that over 95% of the vesicle protein was retained by the Millipore filter. (c) Vesicles that had equilibrated with L-[14C]sorbose were isolated by centrifugation as well as by Millipore filtration. The two methods gave the same value for the amount of sorbose within the vesicles, after correction for the extravesicular space in the pellet through the use of the slowly permeant compound myo-[3H]inositol as a marker. (d) Filters from assays performed at 10% and 100% equilibration of the sorbose were shaken with 0.2 ml of 3 mM L-sorbose/1% Triton X-100. The extracts were subjected to thin layer chromatography on silica gel plates with ethanol/water (4:1 by volume) as the solvent (9). Ninety per cent or more of the radioactivity was recovered at the position of sorbose. Generally this procedure was used to determine the initial rates of uptake by sampling at five time intervals over the period required for about 90% equilibration of the sorbose (see Fig. 3). In order to determine the amount of sorbose taken up at equilibrium, the assay mixtures were subsequently left at 25° for 14 to 18 h and sampled again. The percentage of the total radioactivity that was bound to the filter rose from about 0.5% at zero time to 0.4 to 0.8% during the measurement of the initial rates of uptake. The initial rate of sorbose uptake (micromoles/min) decreased by a factor of 2 when the concentration of proteins was halved and increased by a factor of 2 when the sorbose concentration was doubled. Since the monosaccharide transport system of erythrocytes is inhibited by thioglycollate (13) and so could be inactivated by disulphide formation, one preparation of vesicles was made and assayed in the presence of 1 mM dithiothreitol. Its initial rate of sorbose uptake did not differ significantly from that for vesicles prepared from the same batch of ghosts and assayed in the absence of dithiothreitol. In experiments the uptake of myo-inositol as well as L-sorbose was followed by including 0.5 mM myo-[3H]inositol (10 μCi) in the assay mixture and measuring both the 3H and 14C bound to the filters. Here the quench solution contained 0.50 mM myo-inositol.

Rates of Transport into Whole Cells—The uptake of sorbose by intact erythrocytes was measured by a modification of the procedure described by Levine et al. (10). Erythrocytes were washed at room temperature three times with 6 volumes of 150 mM NaCl/5 mM sodium phosphate, pH 5.0, by suspension and centrifugation. The uptake of sorbose was initiated by mixing an aliquot of L-[14C]sorbose in 100 mM sodium phosphate, pH 5, with a suspension of cells that was equilibrated at the desired temperature in a water bath. The mixtures contained 5.9 mM L-[14C]sorbose in the medium and initially 25% cells (v/v). At various times intervals 1.67-ml aliquots were withdrawn and mixed with 9 ml of ice-cold 1% NaCl, 2 mM HEPES, 1.25 mM KI (stopping solution) in a 12-ml glass centrifuge tube. The cells were sedimented by centrifugation, the supernatant was carefully aspirated, and the centrifuge tube was washed through the addition and removal of 10 ml of stopping solution without disturbing the pellet. After the last traces of supernatant had been taken off with a filter paper wick, the pellet was mixed in a Vortex mixer with a 1 ml of 10% trichloroacetic acid. The precipitated protein was packed by centrifugation, and aliquots of the supernatant were counted.

The initial rate of sorbose uptake was determined at 12.5° by taking six aliquots at 2-min intervals after sorbose addition. The plots of sorbose in the cell pellet against time were linear and rose from about 4% to 12% of the value at equilibrium, which was obtained from aliquots of the assay mixture that were kept at 37° for 3 h before stopping. Duplicate determinations of the rate agreed within ±5% of the average value. For these experiments, the number of cells per ml was carefully measured in order that the initial rates of uptake per mg of membrane protein could be calculated. For the determination of the activation energy for sorbose transport, it proved more convenient to follow the uptake of sorbose by assaying at 2, 5, and 10° over the period required for 90% equilibration. This data gave linear first order plots from which the first order rate constants for equilibration were calculated (see Ref. 10). Other Methods—The number of vesicles per mg of protein was determined by counting both the vesicles and beads in an admixture of vesicles with a known number of 1.0-μm polystyrene beads (Dow Chemical Co.) through the use of a Zeiss Universal microscope (11).

The vesicular volume per mg of protein was calculated from the amount of sorbose within the vesicles at equilibrium. Erythrocytes and ghosts were counted with both a hemacytometer and a ZBI Coulter Counter; the two methods agreed within 5%. SDS-polyacrylamide gel electrophoresis was performed as described by Stock and Kant (5). Protein was routinely measured by the Lowry method with crystallized, lyophilized bovine serum albumin (Sigma) as the standard (12). The protein contents of ghosts and vesicles were also determined by amino acid analysis after hydrolysis for 24 and 48 h in 6 N HCl, 0.1% phenol at 105°, with norleucine as an internal standard for these values were 78% and 89% of those found for ghosts and vesicles, respectively, in the Lowry assay. The values for protein reported herein are given on the basis of amino acid content. Acetyl cholinesterase activity was measured as described by Stock and Kant (5). Phospholipids were extracted with chloroform/methanol (2:1) (13), and total phosphorus in the extract was determined by the method of Bartlett (14).
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FIG. 1. SDS-polyacrylamide gel electrophoresis of erythrocyte ghosts (A) and protein-depleted vesicles (B). The gels contained about 10 μg of protein. They were stained with Coomassie blue and scanned at 550 nm. Bands are designated according to Steck (15); TD marks the location of a piece of wire recording the position of the tracking dye.

TABLE I
Protein composition of depleted vesicles

| Band | Percentage in ghosts | Percentage in vesicles | Ratio
|------|----------------------|------------------------|------|
| 1    | 10.4                 | <0.3                   | <0.03|
| 2    | 13.3                 | <0.3                   | <0.03|
| 3    | 25.9                 | 52.0                   | 2.1  |
| 4.1  | 3.9                  | 0.0                    | 1.3  |
| 4.2  | 4.2                  | 5.4                    | 1.3  |
| 5    | 4.0                  | <0.3                   | <0.08|
| 6    | 3.7                  | <0.3                   | <0.09|
| 7    | 3.2                  | 3.0                    | 0.94 |

"Percentage in vesicles/percentage in ghosts.

The vesicles were found to take up sorbose to an equilibrium level over a period of several hours (Fig. 2). Fig. 3 illustrates data for the initial rates of this uptake. The following findings provide evidence that entry into the vesicles occurs by the monosaccharide transport system.

First, sorbose enters the vesicles at least 10 times more rapidly than the cyclic hexahydroxy compound myo-inositol (Fig. 2). Erythrocytes exhibit the same specificity (16, 17).

Second, an Arrhenius plot of the initial rates of sorbose uptake at the various temperatures (Fig. 3) gives an activation energy of 36 kcal/mol. This value is similar to the value of 39 kcal/mol that we have obtained from the temperature dependence of the first order rate constant for equilibration of sorbose with intact erythrocytes. Our values for this rate constant are 0.91, 2.0, 4.0, 6.9, and 12 × 10⁻³ min⁻¹ at 5.4, 8.3, 10.5, 12.5, and 15.5°, respectively.

Third, α-glucose decreases the initial rate of sorbose uptake (Table II). On the assumption that glucose and sorbose compete for the transport system, the expression for the dissociation constant of glucose (K₈) is:

\[ K₈ = [I]/(Vₒ/Vₐ - 1) \]
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DISCUSSION

The activity of the monosaccharide transport system in sealed erythrocyte ghosts has been shown to be equal to that in intact cells (25–27). This finding indicates that no component of the system is lost upon ghost formation. Our results suggest that the major polypeptides of Bands 1, 2, 5, and 6 are not an obligatory part of the monosaccharide transport system. For example, if Band 1 were required for transport, then the activity of the transport system, expressed per mg of Band 1, would have increased 20-fold, from $5.3 \times 10^{-7}$ liters per min per mg of Band 1 protein in erythrocytes to greater than $10^{-4}$ liters per min per mg of Band 1 protein in the depleted vesicles (see Table I and "Results"). Similar increases in activity, expressed per mg of the specific protein, are demanded for Bands 2, 5, and 6 (see Table I and "Results"). While we cannot exclude the possibility that such an increase in activity might arise because of the simultaneous elution of an inhibitor, we consider this occurrence unlikely. The fact that the specific activity of the transport system in the vesicles averages only 59% of that in erythrocytes should not be taken as evidence against an obligatory role for any of the proteins that are enriched in the vesicles (Table I). The alkali treatment may partially inactivate the system, or the eluted proteins may be necessary for maximal activity.

Our conclusion concerning Bands 1, 2, 5, and 6 agrees with the results of other investigations that have involved different approaches. Carter et al. prepared vesicles from ghosts by treatment of the ghosts with trypsin (28). Vesicles that lacked most of either Bands 1, 2, or 5 or Band 6 were obtained by elution of these proteins from the ghosts before the trypsin treatment. The different vesicle preparations all showed the same net uptake of d-glucose relative to L-glucose in a filtration assay. However, the data indicate that almost complete equilibration of the d-glucose occurred within the time of measurement, so that the relative activities of the monosaccharide system in the different vesicle preparations cannot be estimated from the values for net uptake. Kahlenberg et al. have described a selective binding of d-glucose to ghosts in concentrated ammonium sulfate solution that appears to be a property of the transport system (29). Those binding sites are completely recovered in membrane fragments of ghosts from which most of Bands 1, 2, 5, and 6 have been eluted by treatment with either diiodosalicylate or dimethylmaleic anhydride. 

where $[I]$ is the concentration of glucose, and $V_i$ and $V_o$ are the initial rates in the presence and absence of glucose (20). The value of $V_o/V_i$ in Table II corresponds to a $K_i$ value of 45 mM. Levine and Stein (20) report a $K_i$ value of 19 mM for glucose inhibition of sorbose entry into intact erythrocytes under somewhat different conditions (13°, 150 mM NaCl, 25 mM sodium phosphate, pH 7.4).

Fourth, cytochalasin B and phloretin, which are inhibitors of the monosaccharide transport system, inhibit sorbose uptake into the vesicles (Table II). The $K_i$ values of the compounds as inhibitors of the transport system in intact erythrocytes are 0.5 µM for cytochalasin B at pH 7.4 (21, 22) and 6 µM for phloretin at pH 8 (23). The concentrations that are required to inhibit transport in the vesicles are in approximate agreement with these values. The observation that at 10 µM concentration cytochalasin B reduces the initial rate of uptake to 14% of the control value indicates that in the absence of inhibitor at least 86% of the sorbose enters by the monosaccharide transport system.

Recovery of Transport Activity in Protein-depleted Vesicles – At concentrations of substrate that are substantially less than the half-saturation constant for transport, the kinetic equation for the net flux of substrate into vesicles by way of facilitated diffusion is (24):

$$J = \frac{d[S]}{dt} = k(E) \frac{[S]}{[S]_0 - [S]_i}$$

where the flux $J$ is the net amount of substrate $d[S]_i$ that has entered through an area of membrane in the time period $dt$ (moles per min per mg of membrane protein), $(E)$, is the amount of transport system in that area of membrane (moles per mg of membrane protein), $[S]_0$ and $[S]_i$ are the substrate concentrations outside and inside the vesicles (moles/liter), and $h$ is a composite constant of the transport system (liters min $^{-1}$ mol $^{-1}$). If $[S]_0$ is 10% or less of $[S]_i$, then the initial rate of entry is approximately equal to the entry flux $(k(E)[S]_i)$, and the constant $k(E)$ is given by division of the initial rate by $[S]_i$.

Duplicate determinations of the initial rates of L-sorbose uptake into a known number of intact erythrocytes were made (see under "Experimental Procedures"). Through use of our value of $8.0 \times 10^{-14}$ mg of protein per ghost, we calculate that $k(E)$ for erythrocytes at 12.5° equals $5.3 \times 10^{-7}$ liters per min per mg of ghost protein.

Values of $k(E)$, at 12.5° for seven preparations of vesicles were calculated from plots of 0 to 10% sorbose equilibration like that shown in Fig. 3. The values ranged from 1.9 to 5.0 $\times 10^{-7}$ liters per min per mg of vesicle protein, with $3.1 \times 10^{-7}$ as the average. Thus, the specific activity of the transport system in the vesicles averages 58% of that in the erythrocytes.

It is worth noting that the above analysis may slightly underestimate the activity of the transport system in the vesicles. If the number of transport systems per unit of area membrane is independent of vesicle size, then since the ratio of surface area to internal volume is larger for smaller vesicles, smaller vesicles will reach an $[S]_i$ equal to 10% of $[S]_i$, more rapidly. For spherical vesicles, the ratio of surface area to volume is proportional to the reciprocal of the diameter, and, as a consequence, it can be shown that when 2.0-µm vesicles (the larger size) have reached an $[S]_i$ value that is 10% of $[S]_i$, 0.5-µm vesicles (the smaller size) will have reached an $[S]_i$ value equal to 35% $[S]_i$.

| Inhibitor          | Concentration | Percentage of activity |
|--------------------|---------------|------------------------|
| d-Glucose          | 25 mM         | 64                     |
| Cytochalasin B     | 1 µM          | 92                     |
| Phloretin          | 10 µM         | 14                     |
|                    | 20 µM         | 25                     |

* Initial rate in the presence of inhibitor/initial rate in its absence.


de et al. (34) report a value of $7 \times 10^{-17}$ mg/ghost for the non-hemoglobin, non-lipid weight.
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hydride (30). Finally, Kasahara and Hinkle have been able to reconstitute specific D-glucose transport in sonicated liposomes through the use of a detergent-solubilized extract of ghosts that contains little or no detectable Band 1, 2, 5, or 6 (31). However, since they have not determined the percentage of recovery of the transport activity in the reconstituted system which contains little or no detectable Band 1, 2, 5, or 6 (31). The possibility that this percentage is low and transport is due to a minor component of the extract cannot be excluded.

The protein-depleted vesicles that we have characterized here may prove to be especially useful as a preparation in which to label specifically the protein(s) that constitute(s) the monosaccharide transport system, through reaction either with an affinity-labeling reagent or with a group-specific bound ligand. The less complex protein composition of the protein per vesicle (32, 33). The vesicles containing the transport system would then be separated from the others using some property of this system, such as its affinity for glucose derivatives.

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