Distribution of Glut1 in detergent-resistant membranes (DRMs) and non-DRM domains: effect of treatment with azide

Darrell Rubin1 and Faramarz Ismail-Beigi2

1Department of Pathology and 2Departments of Medicine and of Physiology and Biophysics, Case Western Reserve University, Cleveland, Ohio 44106-4951

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Rubin, Darrell, and Faramarz Ismail-Beigi. Distribution of Glut1 in detergent-resistant membranes (DRMs) and non-DRM domains: effect of treatment with azide. Am J Physiol Cell Physiol 285: C377–C383, 2003. First published April 9, 2003; 10.1152/ajpcell.00060.2003.—We have previously shown that the acute stimulation of glucose transport in Clone 9 cells in response to azide is mediated by activation of Glut1 and that stomatin, a Glut1-binding protein, appears to inhibit Glut1 function. In Clone 9 cells under basal conditions, ∼38% of Glut1, ∼70% of stomatin, and the bulk of caveolin-1 was localized in the detergent-resistant membrane (DRM) fraction; a significant fraction of Glut1 is also present in DRMs of 3T3-Ll fibroblasts and human red blood cells (RBCs). Acute exposure to azide resulted in 40 and 50% decreases in the content of Glut1 in DRMs of Clone 9 cells and 3T3-Ll fibroblasts, respectively, whereas the distribution of stomatin and caveolin-1 in Clone 9 cells remained unchanged. In addition, treatment of Clone 9 cells with azide resulted in a ∼50% decrease in the content of Glut1 in the DRM fraction of plasma membranes. We conclude that 1) a significant fraction of Glut1 is localized in DRMs, and 2) treatment of cells with azide results in a partial redistribution of Glut1 out of the DRM fraction.

stomatin; caveolin-1; transferrin receptor; sucrose density fractionation; lipid raft

GLUT1, a widely expressed member of the facilitative glucose transporter family, is an intrinsic membrane glycoprotein with 12 putative transmembrane domains (19). The transporter is thought to mediate much of the basal, non-insulin-dependent transport of glucose (18, 24). Glut1 expression and function are regulated by a variety of stimuli and agents, including serum, growth factors, transformation, hypoxia, and inhibitors of oxidative phosphorylation, with the latter two being associated with stimulation of 5′-AMP-activated protein kinase (AMPK) (1, 3, 4, 13, 16, 18, 23, 35–38, 48). The enhancement of Glut1-mediated glucose transport in response to inhibition of oxidative phosphorylation is biphasic, with the acute response (∼1–2 h) being mediated entirely by posttranslational mechanisms (13, 36, 37). The acute response in Clone 9 cells does not involve a substantial increase in the abundance of Glut1 in the plasma membrane, and the increase in the Vmax of transport occurs without a change in the affinity of the transporter for glucose or a decrease in the energy of activation of transport (13, 23). These characteristics suggest that the acute stimulation of glucose transport involves activation ("unmasking") of Glut1 transporters preexisting in the plasma membrane.

Results of recent studies in our laboratory suggest that stomatin might be an inhibitory binding partner of Glut1 in Clone 9 cells (49, 50). Stomatin, also known as human erythrocyte protein band 7.2b, is a widely expressed monotopic integral membrane protein (46). Although its function is not clear, it appears to play a role in the regulation of membrane structure, sodium channel activity, and membrane signaling in both mammalian and nematode systems (8, 17, 27, 30, 31, 34, 41–44). Recent reports indicate that stomatin is concentrated in cholesterol-rich microdomains ("lipid rafts") in human red blood cells (RBCs) (31, 32). The presence of stomatin in lipid rafts and its interaction with Glut1 suggested that lipid rafts might play an important role in control of Glut1 function.

Lipid rafts represent a revision of the fluid-mosaic model of cell membranes. It has become clear that the heterogeneous mixture of lipids in the plasma membrane and some intracellular membranes form dynamic domains of closely packed lipids that contain specific proteins and high levels of sphingolipids and cholesterol (5, 22, 39). Lipid rafts have been characterized as a class of membrane substructures that include caveolae, which are morphologically distinct structures that are enriched in cholesterol and caveolin (40). Lipid rafts and caveolae are biochemically defined by their lipid content, low density, and resistance to solubilization in buffers containing non-ionic detergents at low temperatures (4°C); hence, they are identified as low-density, detergent-resistant membrane (DRM) domains.

The reported localization of stomatin in DRMs of human RBCs (31, 32), in conjunction with results of our studies suggesting that the association of stomatin with Glut1 correlates with decreased glucose transport (49, 50), prompted us to pose the following set of questions: 1) Is Glut1 also, in part, concentrated in DRMs in Clone 9 cells? 2) Is stomatin localized to the DRM fraction in cells other than the human RBC? 3) Does...
exposure of cells to inhibitors of oxidative phosphorylation (such as azide) result in a redistribution of stomatin and/or Glut1 between the DRM and non-DRM domains? These and other questions are relevant, especially in light of a recent report indicating that Glut1, but not Glut3, is mostly localized in the DRM fraction in HeLa cells (29). Results of our studies demonstrate that a fraction of cell Glut1 and the bulk of stomatin reside in DRMs under basal conditions and demonstrate that a fraction of cell Glut1 and the bulk of stomatin reside in DRMs under basal conditions and that stimulation of glucose transport in response to azide is associated with a partial redistribution of Glut1, but not of stomatin or caveolin-1, out of the DRM domains.

MATERIALS AND METHODS

Materials. Cell culture reagents were purchased from In-vitrogen (Grand Island, NY), plasticware through Fisher Scientific, and all other chemicals from Sigma Aldrich or Fisher Scientific, unless otherwise noted. Fugene 6 was purchased from Roche (Indianapolis, IN). Sulfo-NHS-SS-biotin reagent and the D, protein assay kit were purchased from Bio-Rad (Hercules, CA). Four-milliliter ultracentrifugation tubes were purchased from Kendro. Antibodies were purchased from the following companies: mouse anti-caveolin-1 from Transduction Laboratories, rabbit anti-Glut1 from Chemicon (Temecula, CA), mouse anti-human transferrin receptor-1 (hTfR1) from Chemicon, and horseradish peroxidase-conjugated anti-rabbit and anti-mouse IgG secondary antibodies from Sigma Aldrich. Mouse anti-human stomatin monoclonal antibody was a gift from Dr. Rainer Prohaska (University of Vienna, Austria). Western blots were developed using the enhanced chemiluminescence kit from Santa Cruz Biotechnology (Santa Cruz, CA). Nitrocellulose membranes were purchased from Schleicher & Schuell (Keene, NH).

Cell culture. Clone 9 cells were maintained in DMEM supplemented with 10% bovine calf serum (vol/vol) and penicillin-streptomycin. Cells were transfected with pcDNA3-human stomatin plasmid (49) or the empty vector, according to the manufacturer's protocol, to enable the detection of stomatin by the available antibody. Stable transfectants were selected with 0.5 mg/ml Geneticin (G418). Surviving clones were harvested using cloning cylinders and maintained in DMEM containing 0.2 mg/ml G418. A clone expressing the human stomatin was employed in these studies. The rate of glucose transport in the chosen clone under basal conditions and following exposure to 5 mM azide was equivalent to nontransfected Clone 9 cells. Culture medium was replaced with fresh medium 16–24 h before initiation of experiments. 3T3-L1 fibroblasts were maintained as described (26).

Fractionation by sucrose density step-gradient centrifugation. Four 10-cm plates of confluent Clone 9 cells or 3T3-L1 fibroblasts were washed three times with ice-cold PBS and then resuspended into 0.5 ml of ice-cold PBS. All subsequent steps were performed at 4°C. Cell pellets were lysed in 410 μl of buffer A (0.5% Triton X-100, 50 mM HEPES, 150 mM NaCl, 5 mM EDTA, 5 mM EGTA, 20 mM sodium pyrophosphate, 1 mM sodium orthovanadate, 20 mM sodium fluoride, and 0.1 μM PMPSF, pH 7.4). Postnuclear lysate (358 μl) was loaded into the bottom of a 4-ml ultracentrifuge tube for the Sorvall TST 60.4 swinging bucket rotor, adjusted to 40% sucrose with 60% sucrose in buffer B (20 mM Tris, 150 mM NaCl, 1 mM EDTA, and 0.1 μM PMPSF, pH 7.4) to a total volume of 1 ml, and then overlaid with 2 ml of 35% sucrose and 1 ml 5% sucrose in buffer B. Samples were centrifuged at an average of 150 k g for 17 h at 4°C. Fractions (500 μl) were collected from the top of the tube. Aliquots (30 μl) of each sample were subjected to SDS-PAGE.

In separate gels, samples of whole cell postnuclear lysates from control and azide-treated cells were also used for Western blotting. The gels were transferred to nitrocellulose membranes, blocked with 5% (wt/vol) powdered milk, and then incubated overnight with the appropriate primary antibodies. After three washes with TBST (0.05% Tween 20 in Tris-buffered saline), membranes were incubated with the appropriate horseradish peroxidase-conjugated secondary antibody, washed with TBST, and developed by enhanced chemiluminescence.

In each experiment, samples from fractions 2 to 9 from control or azide-treated cells were prepared for Western blotting on the same membrane. Moreover, in each experiment and for each antigen, the densities of the appropriate bands in fractions 2 and 3 were added and divided by the total of band densities in all the fractions. The Bio-Rad Gel Doc 1000 system was used to determine band density.

One-step separation of soluble and insoluble material. Fractions were used to prepare cell lysates as described above, using either Triton X-100, Igepal (NP-40), or 3-[3-cholamidopropyl]dimethylammonio]-1-propanesulfonate (CHAPS) (all at 0.5% in buffer A). Lysates were centrifuged at an average of 128 k g for 1 h at 4°C in 500-μl tubes in a Beckman tabletop ultracentrifuge. Supernatants were removed and pellets were resuspended in an equal volume of fresh buffer. Equal volumes of the supernate and resuspended pellet were analyzed.

Detergent-free isolation of DRMs. This method was adapted from a previously described protocol (45). Cells were lysed with 0.5 M sodium carbonate (pH 11) in buffer A without detergent. Lysates were homogenized with 20 strokes of a Teflon Potter-Elvehjem homogenizer. Finally, aliquots of the homogenate were sonicated with a probe-type sonicator (550 Dismembranator, Fisher Scientific) using four 2-s sessions while on ice. All subsequent steps were performed at 4°C. This material was processed as described for isolation and analysis of the DRM.

Isolation of plasma membrane DRMs and their fractionation. The method of cell surface biotinylation was adapted from a previously described protocol (37). Confluent cells in 10-cm plates were rinsed three times with cold PBS and then incubated with 3 ml of cold biotinylation buffer (120 mM NaCl, 30 mM NaHCO3, 5 mM KCl, and 0.1 mg/ml sulfo-NHS-SS-Biotin, pH 8.5) for 30 min while rocking at 4°C. After three washes with 5 ml of stop buffer (140 mM NaCl, 20 mM Tris, and 5 mM KCl, pH 7.5), cells were scraped into 0.5 ml of stop buffer. All subsequent steps were performed at 4°C. Cell pellets were lysed in 400 μl of buffer A. The postnuclear lysate was fractionated in the sucrose-density step gradient as described above. Fractions (300 μl)2, 3, 6, 7, and 8 were incubated with streptavidin-agarose beads overnight at 4°C on a rotisserie shaker; fractions 1, 4, and 5 were excluded because only trace amounts of Glut1 immunoreactivity were present in these samples. The supernatant of each sample was saved, and the beads were washed three times with Tris-buffered saline containing 1 mM EDTA (TBS-EDTA, pH 7.4), and then eluted with 2× Laemmli buffer. Forty percent of the eluate, called the pulldown (pd), and 10% of the supernatant (sup) were fractionated by SDS-PAGE and analyzed. Films were scanned, and the density of the bands, measured using the Bio-Rad Gel Doc 1000 system, was used to calculate the content of Glut1 in each sample. The fraction of cell Glut1 present in plasma membrane
DRMs was calculated by dividing the sum of Glut1 content in the pulldown of fractions 2 plus 3 by the total amount of Glut1 calculated as the sum of Glut1 content in the pulldown and supernate of all fractions. The ratio of Glut1 in plasma membrane DRMs to total cell Glut1 in control and azide-treated cells was corrected by subtracting the ratio of the same from the mock-biotinylated cells prepared in parallel with each experiment.

Preparation of RBC ghosts. RBC ghosts were prepared from freshly drawn blood as previously described (50). The ghosts were resuspended in buffer A containing 1% Triton X-100 and fractionated in a sucrose density gradient. Fractions 1 (top of the gradient) and 9 (pellet) showed no reaction to any of the antibodies employed and are not shown. B: 3T3-L1 fibroblasts were fractionated as above. C: human red blood cell (RBC) ghosts were lysed in buffer containing 1% Triton X-100 before fractionation. Similar results were obtained in at least 4 independent experiments performed on each of the cell types.

Glucose transport. Cytochalasin B-inhibitable glucose transport was measured using 3H-3-O-methylglucose, as previously described (13, 23).

**RESULTS**

Confluent quiescent Clone 9 cells stably transfected to express the human form of stomatin were lysed at 4°C in buffer containing 0.5% Triton X-100 and then subjected to 16 h of ultracentrifugation in a sucrose density gradient (Fig. 1A). In keeping with previous reports that the transferrin receptor is present in the non-DRM fraction and is solubilized under the above conditions (14), the receptor was found in the high-density fractions (fractions 6, 7, and 8). On the other hand, caveolin-1, which is known to be present in caveolae and is not solubilized under the above conditions, was concentrated in the low-density DRM fractions (fractions 2 and 3) (33). Similar to reports in human RBC ghosts (31, 32), we found that the bulk of stomatin is present in the DRM fractions; in repeated experiments, ~70% of stomatin was concentrated in the DRMs. In contrast, Glut1 was distributed in both the DRM and non-DRM fractions, with the fraction of cell Glut1 localized to the DRMs averaging 38 ± 6% (means ± SE, n = 8). A similar distribution of Glut1 was observed employing nontransfected Clone 9 cells or buffers containing 1% Triton X-100 (data not shown). The colocalization of Glut1 and stomatin in the DRMs, and our previous finding of the inhibitory effect of stomatin on Glut1 function (49), raises the possibility that the fraction of Glut1 in this membrane microdomain may be inactive (masked).

We next determined whether the above-noted presence of Glut1 in the DRM fraction is unique to Clone 9 cells. Employing 3T3-L1 fibroblasts and human RBCs, which are known to express Glut1 near exclusively (47, 51, 52), we found Glut1 to be present in DRM fractions of both cells (Fig. 1B for fibroblasts and Fig. 1C for RBCs). The fraction of cell Glut1 localized to the DRM fraction of 3T3-L1 fibroblasts was 29 ± 1%, and for

Statistical analysis. All results are expressed as means ± SE. Two-tailed t-test was used, and a P value of <0.05 was considered significant.
human RBCs, it was 20 ± 3%. In 3T3-L1 fibroblasts, the bulk of caveolin-1 (>95%) was localized in fractions 2 and 3. Similar to the findings in Clone 9 cells, a fraction of Glut1 colocalized with stomatin in DRMs of RBC ghosts; this is presumably the case in 3T3-L1 fibroblasts, although an antibody that recognizes the murine form of stomatin was not available.

The presence of Glut1 in detergent-resistant membrane domains of Clone 9 cells was additionally determined after preparation of cell lysates with buffers containing 0.5% CHAPS or 0.5% NP-40 (Fig. 2). As expected, caveolin-1 remained in the detergent-insoluble fraction, whereas the transferrin receptor was solubilized. Glut1 was present in both fractions employing either of the two additional detergents. However, the fraction of Glut1 remaining in the detergent-resistant fraction (pellet) varied with the detergent used; in two independent experiments employing the above protocol, the percentage of Glut1 in the insoluble fraction averaged 31, 70, and 39% using Triton X-100, CHAPS, and NP-40, respectively. Despite the variability, it is clear that a significant fraction of cell Glut1 is present in the DRM fraction.

In our attempt to verify the presence of Glut1 in DRMs, we employed a fractionation procedure that does not employ detergents (45). In this method, cells are homogenized in 0.5 M Na₂CO₃ pH 11 (which will also remove peripheral membrane proteins) followed by fragmentation of membranes by sonication prior to the sucrose-density gradient centrifugation. Employing this procedure and by varying the extent of sonication in several different experiments, we could not obtain ideal conditions that would lead to the expected partitioning of caveolin-1 and the transferrin receptor. We observed that although the amount of Glut1 in the light fractions decreased with increased sonication, caveolin-1 became increasingly associated with high-density fractions (data not shown). We conclude that the carbonate/sonication procedure cannot be applied in a valid manner in these cells. Nevertheless, it appears that a percentage of cellular Glut1 may be present in the low-density fraction.

The observation that a fraction of Glut1 and the bulk of stomatin reside in DRMs, and considering that stomatin is a Glut1-associated protein that appears to exert an inhibitory effect on Glut1 function (49), led us to test the hypothesis that treatment of cells with azide and the resulting stimulation of glucose transport is associated with a redistribution of Glut1 and/or stomatin from the DRM fraction. In keeping with previous results (13, 36, 37), exposure of Clone 9 cells to 5 mM azide for 90 min resulted in 4.5 ± 0.8-fold stimulation of glucose transport (P < 0.05). Upon exposure to azide for 90 min, we found that the abundance of Glut1 in the

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**Fig. 3.** Effect of azide on the distribution of Glut1, stomatin, transferrin receptor, and caveolin-1 in detergent-soluble and -insoluble fractions. A: transfected Clone 9 cells were treated with diluent or 5 mM azide for 90 min before lysis in Triton X-100 and fractionation. The resulting blots were incubated with the specific antibodies shown. B: in control or azide-treated cells, the densities of bands representing cell Glut1, stomatin, and caveolin-1 present in fractions 2 plus 3 were expressed as a ratio to the total of densities of the appropriate bands in all fractions. The experiment was repeated 5 times, and results were averaged (means ± SE). *P < 0.05.
low-density fraction decreased, whereas the distribution of stomatin and caveolin-1 remained unchanged (Fig. 3A). In repeated experiments, the content of Glut1 in DRMs of Clone 9 cells decreased by ~40% (from 38 to 22% in control and azide-treated cells, respectively), whereas the fractions of stomatin and caveolin-1 in cell fractions 2 and 3 (DRMs) remained unchanged (Fig. 3B). Importantly, the sumtotal of Glut1 content in all the gradient fractions per unit protein was equal in control and azide-treated cells ($P = $ not significant). Moreover, and in keeping with previous results (36, 37), the content of Glut1 in whole cell lysates was equal in control cells and in Clone 9 cells, exposed to 5 mM azide for 90 min. The distribution of transferrin receptor was unaltered by treatment of cells with azide.

To examine the time course of action of azide on Glut1 redistribution and glucose transport, we performed experiments after 30 min of exposure to azide. In three experiments performed after 30 min of exposure to 5 mM azide, the content of Glut1 in the DRM fraction decreased by 25 ± 2% (means ± SE, $P < 0.05$), whereas the distribution of stomatin remained unaltered; the rate of glucose transport increased 2.5 ± 0.5-fold in cells treated with azide for 30 min ($P < 0.05$) (Table 1).

The effect of azide on the abundance of Glut1 in the DRM fraction was also determined in 3T3-L1 fibroblasts. Similar to the finding in Clone 9 cells, the content of Glut1 in the low-density fractions decreased by ~55% from 29 ± 1 to 13 ± 5% in control cells and in cells treated with 5 mM azide for 2 h, respectively ($n = 4; P < 0.05$). In parallel experiments, the rate of glucose transport in fibroblasts increased 2.8 ± 0.5-fold in cells exposed to 5 mM azide for 2 h ($P < 0.05$).

The above results in Clone 9 cells and 3T3-L1 fibroblasts led us to conclude that treatment of cells with azide, and the resulting stimulation of Glut1-mediated glucose transport, is associated with a net decrease in the content of Glut1 in DRMs. It is, hence, possible that the movement of Glut1 away from the relatively low-membrane fluidity of DRMs (10) and the predicted reduction in the association of Glut1 with stomatin might mediate, in part, the activation (unmasking) of Glut1. This possibility, along with the fact that DRM microdomains are present in both the plasma membrane and intracellular membranes (11), prompted studies on the distribution of Glut1 in plasma mem-

Table 1. Effect of exposure of Clone 9 cells to azide on stimulation of glucose transport and the percent decrease of Glut1 in DRMs

<table>
<thead>
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<th>Duration of Exposure, min</th>
<th>Fold Stimulation of Glucose Transport</th>
<th>%Decrease of Glut1 in DRM</th>
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<tbody>
<tr>
<td>30</td>
<td>2.5 ± 0.5*</td>
<td>25 ± 2%</td>
</tr>
<tr>
<td>90</td>
<td>4.5 ± 0.8*</td>
<td>38 ± 11%</td>
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Clone 9 cells were exposed to 5 mM azide for 30 and 90 min. The effect of azide on glucose transport and on the %decrease of Glut1 in the DRM fractions was determined. Values are means ± SE; $n = 3–4$. *$P < 0.05$. DRM, detergent-resistant membrane.

Fig. 4. Effect of azide on the distribution of Glut1 in plasma membrane detergent-resistant membranes (DRMs). A: transfected Clone 9 cells were treated with diluent or 5 mM azide for 90 min before cell surface biotinylation, lysis in Triton X-100, sucrose-density centrifugation, and streptavidin-agarose pulldown. Cells not reacted with the biotinylation reagent were also examined. "pd" represents 40% of the material pulled down by the streptavidin-agarose beads; "sup" represents 10% of the material remaining in the supernate. B: the percentage of total cell Glut1 present in fractions 2 plus 3 in control and azide-treated cells (and corrected for nonspecific pulldown) was determined in parallel. The fractions of the sample used in the "pd" and "sup," as well as the densitometric readings of Glut1 in all fractions, were employed in the calculation of the percentage of cell Glut1 present in the plasma membrane DRMs. The experiment was repeated 3 times, and results were averaged (means ± SE). *$P < 0.05$. 

brane DRMs (PM-DRMs) in control and stimulated cells. In these experiments, cells were first surface-biotinylated (37), and after lysis in Triton X-100 and sucrose-density centrifugation, samples were incubated with streptavidin-agarose beads. This protocol yields an estimate of the content of Glut1 in the PM-DRM of control and stimulated cells, although the value may represent an underestimate because the biotinylation reaction and isolation procedure may be incomplete. Nevertheless, in three independent experiments, after 90 min of exposure to 5 mM azide, the percentage of total cell Glut1 present in the plasma membrane-DRM fraction decreased by 52 ± 12% ($P < 0.05$) from 5.9 to 2.8% in control and azide-treated cells, respectively (Fig. 4, A and B).

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DISCUSSION

We demonstrate the presence of Glut1 in the DRM domain in three different cell types. The results of our studies are consistent with the reported localization of Glut1 in DRMs in HeLa cells and with the presence of Glut4 in DRMs of the plasma membrane of adipocytes (12, 20, 28, 29, 33). Furthermore, we show the redistribution of Glut1 out of this domain upon treatment with azide. These findings raise a number of intriguing questions, including 1) what is the mechanism by which a fraction of cell Glut1 is incorporated and retained in DRMs?, 2) what sequence of events lead to movement of Glut1 out of these domains?, 3) is the movement selective to Glut1?, and 4) to what extent, if any, does the redistribution of Glut1 account for the stimulation of glucose transport? Mechanisms proposed for localization of proteins to DRMs include high affinity of the protein’s transmembrane domains for the sphingolipid- and cholesterol-rich membrane microdomains, anchoring of proteins by glycosphatidylinositol linkage, protein-protein interactions, or coregulation of posttranslational acylation (palmitoylation) (5). Although the mechanism by which Glut1 is associated with the DRMs is not known, we speculate that palmitoylation may be a potential mechanism. We propose this for several reasons, including 1) palmitoylation is reversible and can occur in the time scale of the observed stimulation of glucose transport in response to azide (2, 21), 2) Glut1 contains an ideally positioned cysteine (Cys 207) that can potentially serve as a substrate for palmitoylation (6, 7, 15), and 3) Glut1 has been reported to be palmitoylated (25). Furthermore, protein acyltransferase activity has been localized to the DRM fraction in some cell types, thereby implicating its compartmentalization as a factor in the recruitment of other proteins to this domain (9).

At present, the signals and sequence of events leading to the movement of Glut1 out of DRMs in response to treatment with azide are not known. Likewise, the role of the redistribution of Glut1 in the observed enhancement of glucose transport following inhibition of oxidative phosphorylation remains unresolved. Nevertheless, on the basis of previous observations that stomatin acts as a Glut1-binding protein and appears to have an inhibitory effect on Glut1 function (49, 50), it is tempting to speculate that the movement of Glut1, but not of stomatin, out of the DRMs in response to azide may contribute to “activation” of Glut1. At the present time, the magnitude of this contribution is not known, especially considering the relative change in the content of Glut1 in DRMs compared with the larger increment in the rate of glucose transport. Further studies are necessary to explore the underlying mechanisms and to determine the role of Glut1 redistribution in the stimulation of glucose transport in response to the inhibition of oxidative phosphorylation.

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REFERENCES


GLUT1 AND DETERGENT-RESISTANT MEMBRANES


