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Data Article

Supportive data on the regulation of GLUT4 activity by 3-O-methyl-D-glucose

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ABSTRACT

The data presented in this article are related to the research article entitled "Regulation of GLUT4 activity in myotubes by 3-O-methyl-D-glucose" (Shamni et al., 2017) [1]. These data show that the experimental procedures used to analyze the effects of 3-O-methyl-D-glucose (MeGlc) on the rate of hexose transport into myotubes were valid and controlled. The stimulatory effect of MeGlc was limited to glucose transporter 4 (GLUT4) and was independent of ambient glucose and protein synthesis. Cornish-Bowden kinetic analysis of uptake data revealed that MeGlc attenuated indinavir-induced inhibition of hexose transport in a competitive manner.

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Specifications Table

Subject area	Cell biology
More specific subject area	*Glucose transport regulation*
Type of data	*Text file, figures*
How data was acquired	*[^{3}H]dGlc uptake assay and [^{3}H]MeGlc transport assays*
Data format	*Analyzed*
Experimental factors	*Wild type- and GLUT4myc-expressing L6 cell were used. Primary cultures of bovine vascular endothelial and smooth muscle cell cultures were used as controls for GLUT1 expressing cells.*
Experimental features	*The uptake assay was performed usually in phosphate-buffered saline (PBS) buffer supplemented with 10 μM of deoxy-D-glucose (dGlc) and 37 kBq/mL of [^{3}H]dGlc or 10 μM of MeGlc and 185 kBq/mL of [^{3}H]MeGlc for 5 min at room temperature. The uptake was then terminated and the myotubes were lysed and taken for liquid scintillation counting. The results are given as pmol or nmol of dGlc or MeGlc, respectively, per mg protein, per min.*
Data source location	*Institute for Drug Research, The Hebrew University Faculty of Medicine, Jerusalem, Israel.*
Data accessibility	*The data are available with this article.*

Value of the data

- We have shown that MeGlc augments the intrinsic activity of GLUT4 in myotubes [1]. The data here show that the assays used to analyze the effect of the MeGlc on GLUT4 activity were valid and reproducible and that the experiments were well-controlled.
- The activity of GLUT1, in contrary to GLUT4, was not modified in the presence of MeGlc.
- The effect of MeGlc on GLUT4 was stereospecific.
- MeGlc reduced indinavir-induced inhibition of hexose transport by GLUT4 in a competitive manner.

1. Data

The data presented here are supportive to the data presented in [1] with no duplications or overlap. The data in Fig. 1 show that repetitive exposure of L6 myotubes to MeGlc augmented the rate of hexose transport into L6 myotubes that were maintained at 25 mM glucose. Fig. 2 shows that MeGlc stimulated hexose uptake in L6 myotubes maintained at 5 mM glucose and that these effects were similar to those observed under 25 mM glucose (Fig. 2 in [1]). No such stimulatory effects of MeGlc were evident in GLUT1 expressing vascular cells (Fig. 3). Analyses of [^{3}H]MeGlc transport into wild-type L6 myotubes (Fig. 4) and of [^{3}H] dGlc uptake into L6 myotubes expressing GLUT4*myc* (Figs. 5 and 6) confirm the validity and suitability of the assays used. Inhibition of protein synthesis with cycloheximide did not interfere with MeGlc effects (Fig. 7). MeGlc exerted its effects also in the presence of glucose in the uptake assay (Fig. 8). Unlike MeGlc, its analog 1-α-methylglucose (1-α-MeGlc) failed to modulate the hexose transport system (Fig. 9). Finally, Cornish-Bowden analysis of the uptake data shows that MeGlc attenuated indinavir-induced inhibition of hexose transport in a competitive manner (Fig. 10).