Figure S1. Mutation analysis of GIST-T1 and GIST-T1/IM-R cells. KIT gene mutation in the GIST-T1 and GIST-T1/IM-R cell lines. Sequencing analysis showed that GIST-T1/IM-R cells harbored a secondary *PDGFRA* mutation on exon 12 (c.1701A>G) in addition to a deletion in *KIT* exon 11. PDGFRA, platelet-derived growth factor receptor α; IM-R, imatinib-resistant.

Cell line	Gene mutation	
	Primary	Secondary
GIST-T1	KIT exon11 (del)	–
GIST-T1/IM-R	KIT exon11 (del)	PDGFRA exon12 (c.1701A>G)

Figure S2. Effects of imatinib administration on the phosphorylation of KIT, AKT and ERK1/2 in the tyrosine kinase receptor cascade in GIST-T1 and GIST-T1/IM-R cells. Quantitative analysis of the phosphorylation levels of the proteins from western blotting (Fig. 1B) for determining the effect of imatinib administration on the tyrosine kinase receptor cascade activity in GIST-T1 and GIST-T1/IM-R cells at 72 h after imatinib treatment (0, 6.5, 13 and 500 nM). Data are expressed as fold change over control. *P<0.05 and ***P<0.001 vs. 0. GIST-T1/IM-R, imatinib-resistant GIST-T1 cells.

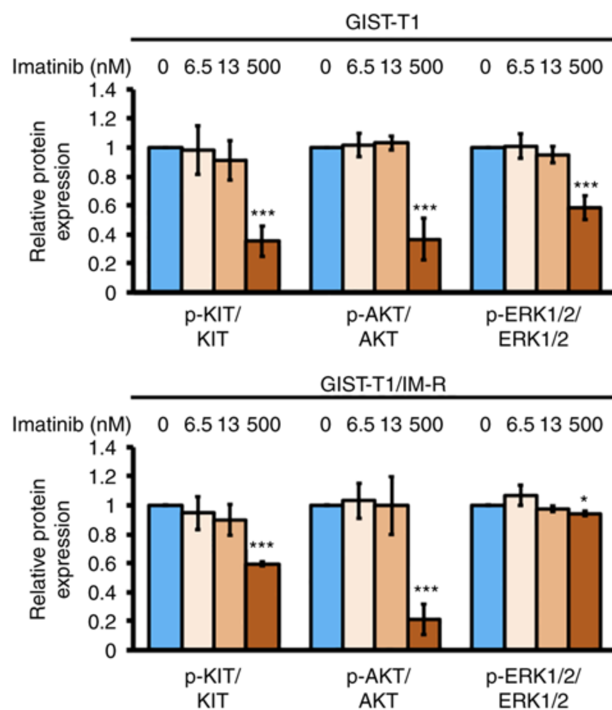

Figure S3. Expression of glycolysis-related proteins in GIST-T1 and GIST-T1/IM-R cells after imatinib treatment. Quantitative analysis of the expression and/or phosphorylation levels of the glycolysis-related proteins GLUT-1, HK2, PKM2 and LDHA from western blotting (Fig. 2B) for determining the effects of imatinib administration. Imatinib (0, 6.5 and 13 nM) was administered to both GIST-T1 and GIST-T1/IM-R cells. The effect was examined at 72 h after imatinib treatment. Data are expressed as fold change over control. *P<0.05, **P<0.01 and ***P<0.001 vs. 0. GIST-T1/IM-R, imatinib-resistant GIST1-T1 cells; GLUT-1, glucose transporter; HK2, hexokinase 2; p-, phosphorylated; PKM2, pyruvate kinase M2; LDHA, lactate dehydrogenase.

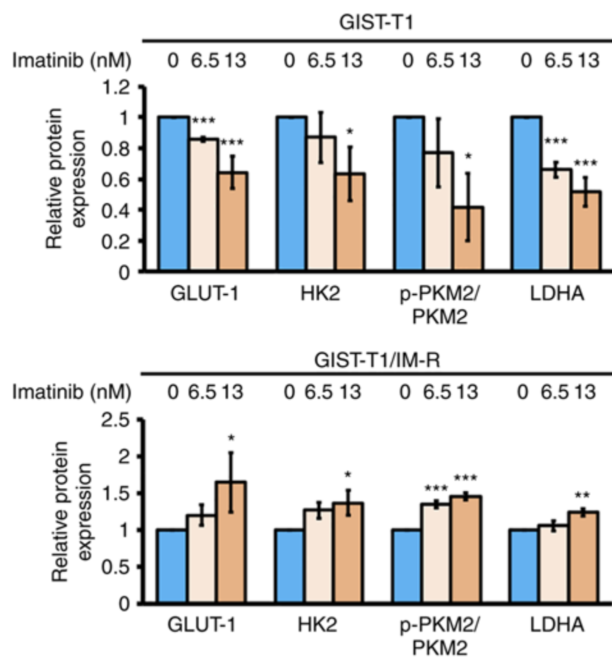

Figure S4. Expression of glycolysis-related proteins in GIST-T1/IM-R cells after transfection with siR-*SLC2A1* and/or imatinib administration. Quantitative analysis of the expression and/or phosphorylation levels of glycolysis-related proteins HK2, PKM2 and LDHA in the western blotting images (Fig. 3D) at 72 h after treatment with imatinib (13 μM) and/or siR-*SLC2A1* (10 nM) transfection. Data are expressed as fold change over control. *P<0.05 vs. Control. siR, small interfering RNA; SLC2A1, solute carrier family 2 member 1; GIST-T1/IM-R, imatinib-resistant GIST1-T1 cells; HK2, hexokinase 2; p-, phosphorylated; PKM2, pyruvate kinase M2; LDHA, lactate dehydrogenase.

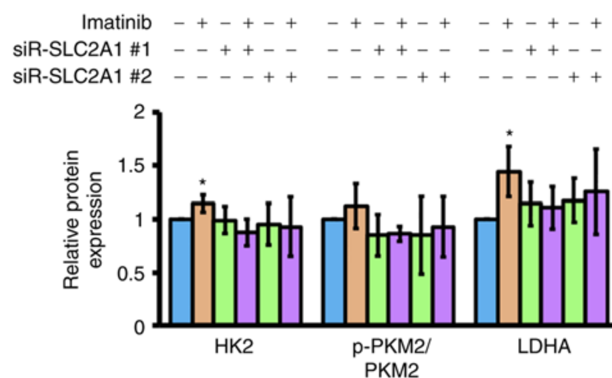

Figure S5. Effect of glucose transporter-1 inhibitor WZB117 administration on the viability of GIST-T1/IM-R cells. IC_{50} of WZB117 in GIST-T1/IM-R cells as determined by MTT assay 72 h after WZB117 treatment. The IC_{50} of WZB117 was 15.8 μM in GIST-T1/IM-R cells. GIST-T1/IM-R, imatinib-resistant GIST1-T1 cells; IC_{50}, half-maximal inhibitory concentration.

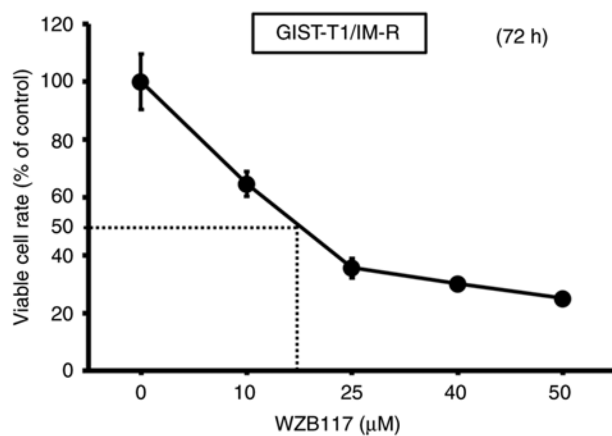

Figure S6. Effects of treatment with the GLUT-1 inhibitor WZB117 and/or imatinib on the expression of glycolysis-related proteins in GIST-T1/IM-R cells. (A) Quantitative analysis of the expression and/or phosphorylation levels of glycolysis-related proteins GLUT-1, HK2, PKM2 and LDHA after treatment of GIST-T1/IM-R cells with WZB117 (10 μM) for various time periods (0 to 72 h) (Fig. 4C). (B) Quantitative analysis of the expression and/or phosphorylation levels of glycolysis-related proteins GLUT-1, HK2, PKM2 and LDHA in GIST-T1/IM-R cells at 6 h after treatment with imatinib (13 nM) and/or WZB117 (10 μM) (Fig. 4D). Data are expressed as fold change over control. *P<0.05; **P<0.01 and ***P<0.001 vs. 0 or Control. $^{\#}$P<0.05. GIST-T1/IM-R, imatinib-resistant GIST-T1 cells; GLUT-1, glucose transporter 1; HK2, hexokinase 2; p-, phosphorylated; PKM2, pyruvate kinase M2; LDHA, lactate dehydrogenase.

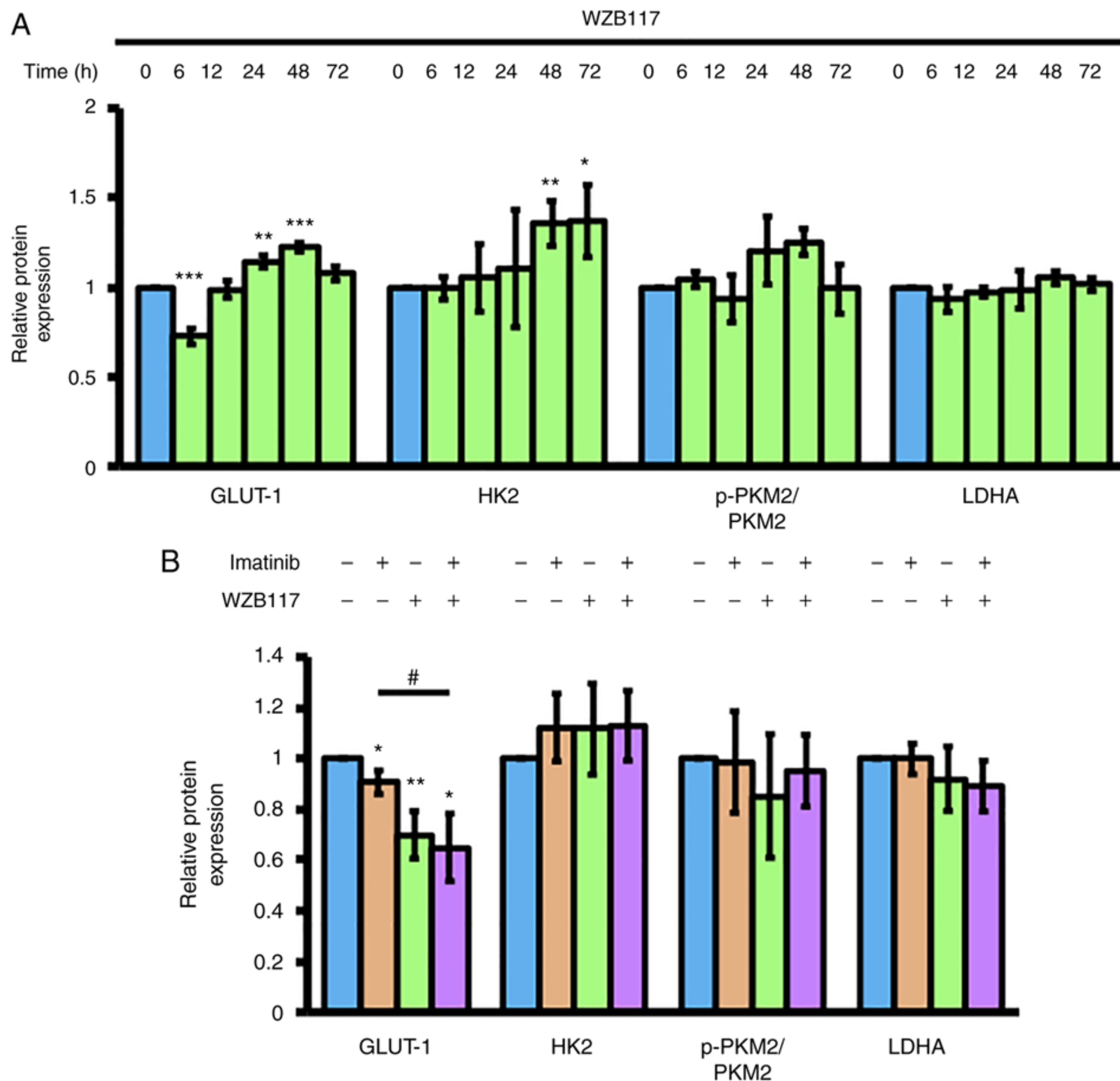

Figure S7. Expression of proteins in the growth/survival and apoptosis signaling pathways after *SLC2A1* knockdown and/or imatinib treatment in GIST-T1/IM-R cells. Quantitative analysis of the expression levels of cell growth/survival signaling and apoptosis-related proteins at 72 h after treatment with imatinib (13 nM) and/or siR-*SLC2A1* (10 nM) transfection in GIST-T1/IM-R cells (Fig. 5A). Data are expressed as fold change over control. Each value is in comparison to the control. $^{**}P<0.01$ and $^{***}P<0.001$ vs. Control. $^{\#}P<0.05$ and $^{\#\#\#}P<0.001$. siR, small interfering RNA; SLC2A, solute carrier family 2 member 1; p-, phoshorylated; PARP, poly (ADP ribose) polymerase; casp, caspase; n.s., not significant.

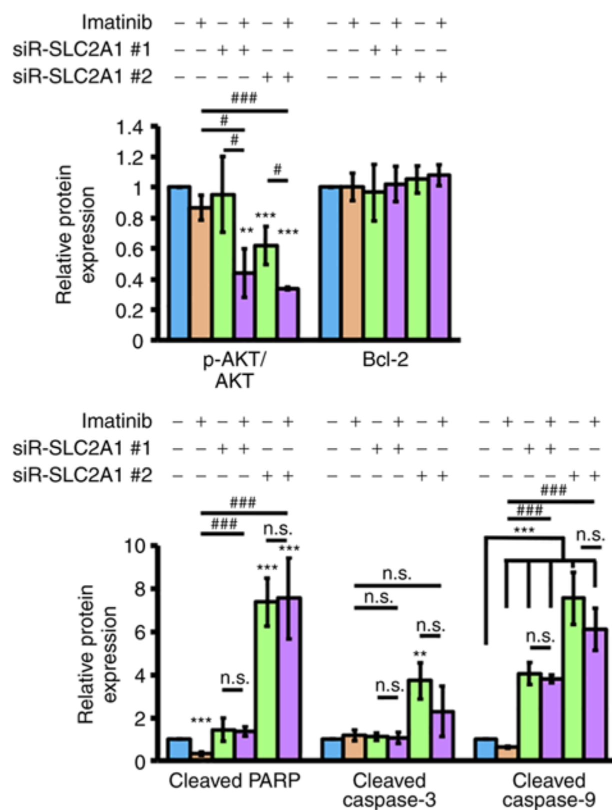

Figure S8. Expression of growth/survival signaling and apoptosis-related proteins after treatment with the glucose transporter-1 inhibitor WZB117 and/or imatinib in GIST-T1/IM-R cells. Quantitative analysis of the expression levels of cell growth/survival signaling and apoptosis-related proteins in GIST-T1/IM-R cells at 6, 12 and 72 h after treatment with imatinib (13 nM) and/or WZB117 (10 μM) (Fig. 6A). Data are expressed as fold change over control. *P<0.05; **P<0.01 and ***P<0.001 vs. Control. #P<0.05, ##P<0.01 and ###P<0.001. p-,phosphorylated; PARP, poly (ADP ribose) polymerase; casp, caspase; n.s., not significant.