



GLUT4 *endocytosis* requires PtdIns(4,5)P₂, whose insulin-dependent regulation is uncertain. A subpopulation of GLUT4 *exits* SE/EE (sorting endosomes/early endosomes) and *via* RE/TGN (recycling endosomes/trans-Golgi network) *enters* into GSVs (GLUT4 storage vesicles) in an insulin-regulated process that requires robust endosomal PtdIns(3,5)P₂ synthesis by ArPIKfyve/PIKfyve. A subpopulation of small GLUT4 vesicle intermediates that may also bud off from SE/EE contain PtdIns4K IIα and remain insulin-unresponsive (NRC). Insulin-dependent *movement* of GSV requires PI3K-C2α-catalyzed PtdIns(3)P synthesis that may also affect events at the cells surface. GLUT4 *movement/tethering* requires an insulin-regulated F-actin remodeling, regulated by robust change in PtdIns(5)P- and/or PtdIns(4,5)P₂. Signals mediated by insulin-activated class IA PI3K and PtdIns(3,4,5)P₃ synthesis regulate GLUT4 *docking/fusion*. The PtdIns(3,4,5)P₃ (red), PtdIns(3)P (yellow) and PtdIns(4,5)P₂ (orange) basal or insulin-regulated localizations at plasma membrane subdomains are confirmed by specific reporters, containing selective PH domains [for PtdIns(3,4,5)P₃ and PtdIns(4,5)P₂ binding] or the FYVE finger domain [for PtdIns(3)P binding]. Compartments/microdomains of basal or altered PtdIns(3,5)P₂, PtdIns(5)P and PtdIns(4)P in response to insulin have not been verified by specific bioreporters. The individual steps in the overall GLUT4 translocation are in italics.