Title:	Focal Adhesion Kinase contributes to insulin-induced actin reorganization into a mesh harboring glucose transporter-4 in insulin resistant skeletal muscle cells
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Abstract:	Background: Focal Adhesion Kinase (FAK) is recently reported to regulate insulin resistance by regulating glucose uptake in C2C12 skeletal muscle cells. However, the underlying mechanism for FAK-mediated glucose transporter-4 translocation (Glut-4), responsible for glucose uptake, remains unknown. Recently actin remodeling was reported to be essential for Glut-4 translocation. Therefore, we investigated whether FAK contributes to insulin-induced actin remodeling and harbor Glut-4 for glucose transport and whether downregulation of FAK affects the remodeling and causes insulin resistance. Results: To address the issue we employed two approaches: gain of function by overexpressing FAK and loss of function by siRNA-mediated silencing of FAK. We observed that overexpression of FAK induces actin remodeling in skeletal muscle cells in presence of insulin. Concomitant to this Glut-4 molecules were also observed to be present in the vicinity of remodeled actin, as indicated by the colocalization studies. FAK-mediated actin remodeling resulted into subsequent glucose uptake via PI3K-dependent pathway. On the other hand FAK silencing reduced actin remodeling affecting Glut-4 translocation resulting into insulin resistance. Conclusion: The data confirms that FAK regulates glucose uptake through actin reorganization in skeletal muscle. FAK overexpression supports actin remodeling and subsequent glucose uptake in a PI3K dependent manner. Inhibition of FAK prevents insulin-stimulated remodeling of actin filaments resulting into decreased Glut-4 translocation and glucose uptake generating insulin resistance. To our knowledge this is the first study relating FAK, actin remodeling, Glut-4 translocation and glucose uptake and their interrelationship in generating insulin resistance.
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