

AKT and pAKT Expression in Different Insulin Sensitive Tissues from C57BL/6J Mice

Siva, Nanda

Purpose: Type II diabetes, along with obesity increasing in prominence in the United States and other countries, is characterized by hyperglycemia and insulin resistance to glucose uptake in peripheral tissues. Obesity plays a role in the induction of this insulin resistance. AKT (also known as protein kinase B [PKB]) has been shown to function in the insulin receptor-signaling cascade. AKT is activated through its phosphorylation which triggers an intracellular downstream signaling processes that leads to glucose uptake into target cells. The goal of this project was to analyze the level of expression of AKT and phosphorylated AKT (pAKT) in different insulin sensitive tissues of obese and normal mice. **Procedure:** For this purpose, extraction was performed from liver, muscle and mesenteric fat samples, which had already been collected from C57BL/6J mice that were fed Low Fat Diet (normal) or High Fat Diet (obese) for 31 weeks. To assess the effects of the HFD/obesity on basal and insulin stimulated AKT phosphorylation, protein samples from the three tissues were subjected to denaturing electrophoresis. AKT and pAKT expressions were analyzed using specific antibodies by the Western Blot technique. **Data:** All tissues displayed basal expression of AKT; however, HFD treated animals showed lowers level of insulin stimulation compared to the LFD. For example, average ratios of pAKT (+/-) insulin of 11.47(LFD)/3.98 (HFD) were seen in liver. Muscle and mesenteric fat displayed a similar trend. **Conclusion:** These results suggest that ratios of pAKT/AKT expression can be used as a method to assess insulin resistance.

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