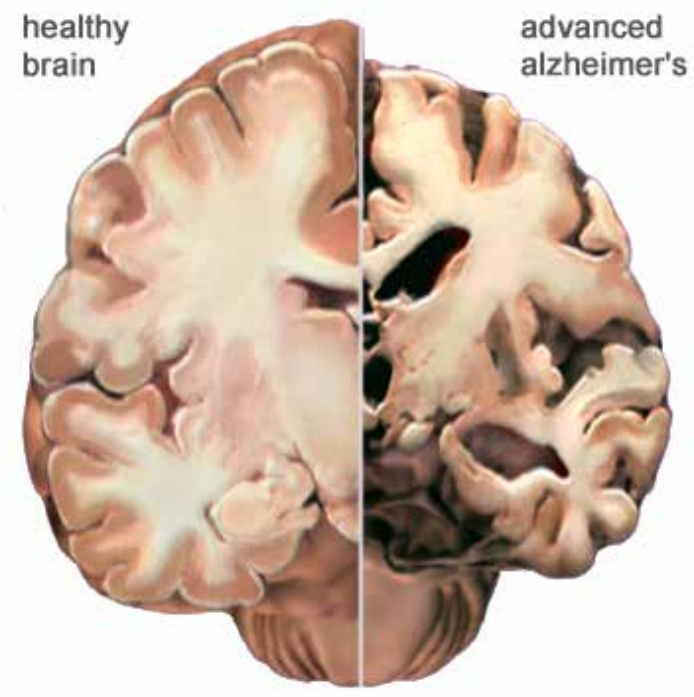
Linking Alzheimer's Disease and Insulin Signaling

Research has indicated a potential link between Alzheimer's disease (AD) and insulin resistance for several years. Commonalities between AD and type 2 diabetes mellitus (T2DM) are highlighted by interruptions in insulin signaling that affect cell growth, cell differentiation, cellular repair mechanisms, energy metabolism, and glucose utilization[1]. Insulin is primarily known for regulating blood sugar levels but also plays a significant role in neuronal function as it acts as a growth factor for both regular cells as well as neurons.

Recent research highlights links between AD and increased resistance to insulin in the brain. Insulin resistance appears to be an early, common feature of AD and is accompanied by insulin-like growth factor (IGF) resistance[2]. This resistance develops in the brains of subjects with AD even without the presence of T2DM.

By restoring insulin responsiveness and lowering insulin depletion in the brain, studies suggest that it may be possible to slow or halt the effects of AD. Pharmacological agents that can perform these functions are currently under investigation and are being studied for their effects in T2DM models. **Metformin Hydrochloride (M2076)**is a commonly used treatment for T2DM.



Metformin is an insulin sensitizer that decreases hepatic glucose production. In pre-clinical studies, metformin sensitizes neuronal insulin resistance[3].

LKT Laboratories carries a variety of antidiabetic compounds. Other potentially useful antidiabetic therapies include PPAR agonists **Pioglitazone Hydrochloride (P6954)** and **Troglitazone (T7056)** and ATP-sensitive K^+ channel modulators **Glimepiride (G4535)** and **Repaglinide (R1860)**.

References:
1. Gao C, Liu Y, Li L, et al. Rev Neurosci. 2013;24(6):607-15.
2. Talbot K, Wang HY, Kazi H, et al. J Clin Invest. 2012 Apr;122(4):1316-38.
3. Gupta A, Bisht B, Dey CS. Neuropharmacology. 2011 May;60(6):910-20.