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## The role of lysophosphatidic acid receptor signaling in diabetic nephropathy in db/db mice

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Diabetic nephropathy is the major microvascular complication of both type-1 and type-2 diabetes. Lysophosphatidic acid (LPA) is a small, ubiquitous phospholipid involved in cellular processes such as proliferation, survival, migration and suppression of apoptosis by binding to LPA receptors (LPARs). Previously, it was reported that serum level of LPA is elevated in the diabetic conditions, but the involvement of LPA in development of diabetes and its complications remains unknown. We investigated the role of LPA signaling in diabetic nephropathy and the molecular mechanisms involved. The mRNA level of LPAR1 was significantly increased in both high-glucose maintained mesangial cells (SV40 MES13) and the kidney cortex of diabetic db/db mice. Increased albuminuria, serum creatinine, glomerular tuft area and glomerular volume were observed in db/db mice, this was reduced by LPAR1 antagonist (ki16425) treatment. TGFβ expression was up-regulated in SV40 MES13 cells by LPA stimulation or in the kidney cortex of db/db mice, but which were blocked by ki16425 treatment. Further studies showed that LPA treatment of SV40 MES13 cells increased phosphorylated glycogen synthase kinase (GSK) 3-β at ser9 and induced translocation of sterol regulatory element-binding protein (SREBP1) into the nucleus. Blocking phosphorylation of GSK3β inhibited SREBP1 activation and consequently blocked LPA-induced TGF-β expression in SV40 MES13 cells. These results suggest that glycogen synthase kinase (GSK) 3-β (Ser9) phosphorylation and subsequent sterol regulatory element-binding protein (SREBP) 1 activation is involved in TGFβ production induced by LPA in the diabetic condition.

### Biography

Yongha Hwang is currently pursuing Master's degree at College of Pharmacy, Gachon University, South Korea. His research interests focus on the development of therapeutic intervention for treatment of diabetes.

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