Diabetic nephropathy (DN) is a diabetic vascular complication, and abnormal protein kinase C (PKC) activation from increased diacylglycerol (DG) production in diabetic hyperglycemia is one of the causes of DN. Diacylglycerol kinase (DGK) converts DG into phosphatidic acid. In other words, DGK can attenuate PKC activity by reducing the amount of DG. Recently, we reported that intraperitoneally administered d-α-tocopherol (vitamin E, αToc) induces an amelioration of DN *in vivo* through the activation of DGKα and the prevention of podocyte loss. However, the effect of the oral administration of αToc on DN in mice remains unknown. Here, we evaluated the effect of oral administration of αToc on DN and its molecular mechanism using streptozocin-induced diabetic mice. Consequently, the oral administration of αToc significantly ameliorated the symptoms of DN by preventing the loss of podocytes, and it was revealed that the inhibition of PKC activity was involved in this amelioration.

The assumed mechanisms of the protective effect of d-α-tocopherol on diabetic nephropathy.