Palmitoylation of 67kDa laminin receptor

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Diacylglycerol kinase (DGK) is a lipid kinase to convert DG to phosphatidic acid (PA). Both DG and PA are important lipid messengers. Thereby, DGK is thought to have important roles in lipid signaling. In addition, the enzyme recently has been gotten attention as a pharmaceutical target because of its involvement in cancer, neuronal disease and diabetes etc. We have reported that Epigallocatechin gallate (EGCg) and α -tocopherol (VtE) activate DGK α via 67kDa laminin receptor (67LR), resulting in improvement of diabetic nephropathy. The 67LR is believed to be concentrated in lipid raft as homo dimer. However, it is still unknown whether 67LR is palmitoylated although many proteins localized in raft have the modification. Therefore, we investigated palmitoylation of 67LR and its physiological meaning in the activation of DGK α .

67LR was slightly palmitoylated in resting state. Both EGCg and VtE treatment induced palmitoylation. The VtE-induced palmitoylation was peaked at 5 min and then decreased gradually. The time course of the palmitoylation of 67LR was fit to a time course of translocation (activation) of DGK α by VtE. The VtE-induced translocation of DGK α was abolished by treatment of a palmitoyltransferase inhibitor. These results indicate the palmitoylation of 67LR is necessary for the activation of DGK α by VtE or EGCg. The physiological meaning of the palmitoylation of 67LR in localization and dimerization are under investigation.