Oleic acid-induced PKC isozyme translocation in RAW 264.7 macrophages (In press)

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摘要

Abstract

Fatty acids are important second messengers that mediate various cellular functions, but their role in the formation of macrophage foam cells is not known. High plasma levels of oleic acid (OA) in obese patients are often associated with a high risk for atherosclerosis. In this study, we investigated the protein kinase C (PKC) isozymes involved in OA-induced lipid accumulation in RAW 264.7 macrophages. The results show that OA induces translocation of PKC alpha, beta1, and delta from the cytosol to the cell membrane 5 min after the treatment. After 16 h incubation with OA, PKC delta was found to be colocalized with adipose differentiation-related protein (ADRP) on the surface of lipid droplets, but immunoprecipitation experiments showed that PKC delta was not biochemically associated with ADRP. After 16 h incubation with OA plus phorbol 12-myristate 13-acetate (PMA), PKC delta staining on the lipid droplet surface was not seen, whereas the accumulation of lipid droplets was unaffected. Furthermore, downregulation of PKC delta was confirmed by immunoblotting. These results demonstrate possible involvement of specific PKC isozymes in the early phase of lipid accumulation, possibly during the uptake of OA.