Immunocytochemical studies on lipid droplet-surface proteins in adrenal cells

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摘要

Abstract

Perilipin and ADRP, located on the surface of intracellular lipid droplets, are proposed to be involved in adipocyte lipid metabolism. The aim of the present study was to investigate the effect of PKA and PKC activities on the distribution of perilipin and ADRP in primary cultured adrenal cells, and the role of ERK in PMA- and calphostin C-induced steroidogenesis. Immunofluorescence staining indicated that in addition to p160, a capsular protein of steroidogenic lipid droplets, perilipin and ADRP were localized on the lipid droplet surface. Stimuli such as activation of PKA by db cAMP or inhibition of PKC by calphostin C, which increase corticosterone synthesis in various magnitudes, caused detachment of p160 and perilipin, but not ADRP, from the lipid droplet surface. Activation of PKC by PMA induced increase in corticosterone synthesis, however, it did not affect the distribution of perilipin, p160, or ADRP on the lipid droplet surface, suggesting the presence of mechanisms for promoting sterodiogensis other than causing detachment of lipid droplet surface proteins. We further demonstrated that ERK pathway was involved in PMA-induced steroidogenesis, since PD98059, specific inhibitor of MEK, blocked the increases in steroidogenesis and phosphorylation of ERK caused by PMA, but not by cAMP-PKA. These data indicate that p160, perilipin, and ADRP were all located on the lipid droplet surface in rat adrenal cells. On the basis of its non-responsiveness to lipolytic stimulation, ADRP may be a structural protein of the lipid droplet surface, whereas their immediate response to lipolytic stimuli suggest that perilipin and p160 are functional proteins. PKC regulates adrenal steroidogenesis through ERK cascade, whereas PKA pathway does not involve ERK.