Lysophosphatidylcholine (LPC) induces apoptosis via G2A receptor-dependent pathway in hippocampal progenitor cells

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Lysophosphatidylcholine (LPC) is a bioactive lipid molecule involved in numerous biological processes. G2A is a G protein-coupled receptor for which LPC is a high affinity ligand. Previous studies demonstrated that LPC induces mitogen-activated protein kinases (MAPK) in cultured cells. However, the role of LPC-activated protein kinases in neural cells remains undefined. In this study, we examined the effect of LPC on apoptosis and investigated the role of G2A and LPC-activated protein kinases in rat hippocampal progenitor cells (H19-7). We found that LPC strongly induced apoptosis in a time- and dose-dependent manner, as revealed by morphological criteria, cytotoxicity assay, DNA fragmentation assay, and TUNEL analysis. LPC stimulated the phosphorylation of ERK1/2 and JNK/SAPK in H19-7 cells. Using the anti-G2A antibody, we found that G2A was specifically required for LPC-induced apoptosis. Furthermore, LPC-induced apoptosis was inhibited by DEVE-fmk (a caspase-3/CPP32 inhibitor), suggesting involvement of an important segment in the apoptosis. These results demonstrate that G2A/LPC-induced apoptosis involves caspase-3 mediated pathways in H19-7 cells.