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Lysophosphatidylcholine-induced surface redistribution regulates signaling of the murine G-protein-coupled receptor G2A

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Intracellular trafficking and spatial dynamics of membrane receptors critically regulate receptor function. Using microscopic and subcellular fractionation analysis, we studied the localization of the murine G-protein-coupled receptor G2A (muG2A). Evaluating GFP tagged-exogenously expressed, as well as the endogenous muG2A, we observed that this receptor was spontaneously internalized and accumulated in endosomal compartments, while its surface expression was enhanced and stabilized by LPC treatment. Other members of this GPCR family can be activated by protons. Exposure to pH ranging from 6.2 to 7.8 had no effect on the localization of muG2A in the absence or presence of added LPC. Monensin, a general inhibitor of recycling pathways, blocked LPC-regulated surface localization of muG2A, as well as muG2A-dependent ERK activation and cell migration induced by LPC treatment. Mutation of the conserved DRY motif (R•A) enhanced the surface expression of muG2A, resulting in its resistance to monensin inhibition of ERK activation. Our data suggest that intracellular sequestration and surface expression regulated by LPC, rather than direct agonistic activity control the signaling responses of murine G2A towards LPC.