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## POSTER ABSTRACT - 2005

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### G2A signaling in lysophosphatidylcholine induced cell migration

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G2A is a G protein-coupled receptor predominantly expressed in hematopoietic cells. We here show that the chemotaxis of mouse macrophages and T cells to lysophosphatidylcholine (LPC) is dependent on G2A expression. The LPC/G2A interaction may be very complex since recent results suggest LPC is not likely a direct ligand for G2A. Interestingly, the LPC/G2A-mediated chemotaxis is resistant to pertussis toxin treatment, suggesting Gi/o is not required for the process. Inhibition with dominant negative G proteins and RGS constructs indicates that Gq/11 and G12/13 are involved. To further delineate downstream signaling important for LPC/G2A-mediated cell migration, we developed a chemotaxis assay to screen the Biomol small molecule libraries of inhibitors for kinases, phosphatases and ion channels. Treatment of DO11.10 cells with inhibitors for phosphatidylinositol 3-kinase, protein kinase C, calcineurin, and calcium channels significantly abolished the LPC/G2A induced cell migration, suggesting these pathways are important for this process. We also studied the structure-activity relationship of LPC-related lysolipids in G2A-mediated J774 macrophage chemotaxis. Lyso platelet activating factor and lysophosphatidylglycerol exhibited weaker chemotactic activity through G2A, but other lysolipids such as lysophosphatidylinositol, lysophosphatidylserine, lysophosphatidylethanolamine, lysophosphatidic acid and sphingosylphosphorylcholine showed no chemotactic activity or G2A-independent activity in J774 cells. These results suggest the specificity of lysolipids on G2A-mediated cell migration and argue against that the cone-shape effect of lysolipids is the cause of cell migration in our system.