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Abstract
Monocyte and macrophage are key players in atherosclerosis. In response to chemokines and cytokines released by inflammatory stimuli, monocyte adheres with vascular endothelium and migrates through the vascular wall. Fractalkine, a CX3C chemokine expressed in endothelial cells, is involved in activation and adhesion of monocyte. Interaction of fractalkine with its receptor CX3CR1 stimulates a variety of protein kinases, such as MAPK, Src, and PI 3-kinase. However, the role of hematopoitic-specific tyrosine kinases and Rho family in fractalkine signaling remains to be elucidated. In addition, it is not clear regarding the fractalkine's effect on membrane rafts, which are specialized microdomains of plasma membrane characterized by enrichment of cholesterol, sphingolipids, and signaling molecules. In this study, we showed in THP-1 monocytic cells that fractalkine stimulates an increase of protein tyrosine phosphorylation and activation of Lyn and ERK2, but not JNK and p38 MAPK. Our results also indicated that fractalkine induces activation of Rho A, an important regulator of cytoskeleton, and re-organization of F-actin. It is of interest to note that dynamic changes of membrane rafts components are observed in fractalkine-treated monocytes, as evidenced by the change of distribution for flotillin-2, a rafts marker, and signaling molecules G i-2, Lyn and Rho A. These findings implicate an involvement of membrane rafts and coupling of G i-2, Lyn and RhoA in fractalkine signaling in monocytes. These signaling events may lead to cytoskeleton reorganization and play an important role in monocyte migration in response to fractalkine stimulation.
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