FEATURES

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Dynamics of Focal Adhesions and Reorganization of F-Actin in VEGF-Stimulated NSCs Under Varying Differentiation States

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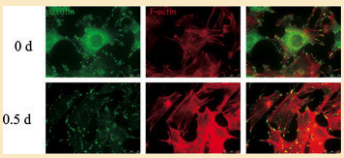

Precise migration of neural stem/progenitor cells (NSCs) is crucially important for neurogenesis and repair in the nervous system. However, the detailed mechanisms are not clear. Our previous results showed that NSCs in varying differentiation states possess different migratory ability to vascular endothelial growth factor (VEGF). In this study, the different dynamics of focal adhesions (FAs) and reorganization of F-actin in NSCs during spreading and migration stimulated by VEGF. VEGF is demonstrated. It is found that the migrating NSCs of 0.5 d and 1 d differentiation possess more FAs at leading edge than cells of other states. Moreover, the phosphorylation of focal adhesion kinase (FAK) and paxillin in NSCs correlates closely with their differentiation states. VEGF promotes FA formation with broad lamellipodium generation at the leading edge in chemotaxing cells of 0 d, 0.5 d and 1 d differentiation, but not in cells of 3 d differentiation. Furthermore, cells of 1 d differentiation show a maximal asymmetry of FAs between lamella and cell rear, orchestrating cell polarization and directional migration. Time-lapse video analysis shows that the disassembly of FAs and the cell tail detachment in NSCs of 1 d differentiation are more rapid, along with the concurrent enlarged size of FAs at the leading edge, leading to the most effective chemotactic response to VEGF. Collectively, these results indicate that the dynamics of FAs and reorganization of F-actin in NSCs that undergo directional migration correlate closely with their differentiation states, contributing to the different chemotactic responses of these cells to VEGF.

The Suppressive Role of p38 MAPK in Cellular Vacuole Formation

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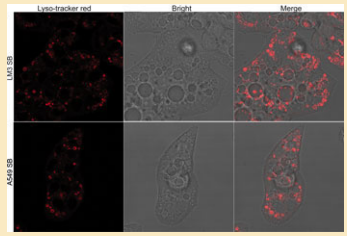

Vacuolization of the cytoplasm is one of the dramatic and frequently observed phenomena in various cell types. Cellular vacuoles occur spontaneously or via a wide range of inductive stimuli, but the molecular mechanism involved in this process remains largely unknown. In this study, the role of the p38 and JNK pathways in the formation of cytoplasmic vacuoles is investigated. It is found that p38 and JNK agonist anisomycin abolishes spontaneous cytoplasmic vacuolization of HepG2 cells through p38 activation, but not through JNK activation. Importantly, blocking the activity of p38 or suppression the expression of p38 elicits cytoplasmic vacuoles formation in various cancer cells. Furthermore, cytoplasmic vacuoles induced by p38 blocking are derived from the perinuclear region. These observations provide direct evidence for a role of p38 signaling in regulating the formation of cytoplasmic vacuoles.