
We have examined the function of a member of the vasodilator–stimulated phosphoprotein family of proteins (DdVASP) in Dictyostelium. Ddvasp null cells lack filopodia, whereas targeting DdVASP to the plasma membrane with a myristoyl tag results in a significant increase in filopodia. The proline–rich domain–Ena/VASP homology 2 structure is required for both actin polymerization activity and filopodia formation. Ddvasp null cells exhibit a chemotaxis defect, which appears to be due to a defect in the ability of cells to properly adhere to the substratum and remains elevated for up to 1 min. These defects lead to a significant decrease in chemotaxis efficiency. DdVASP localizes to the leading edge in migrating cells and to the tips of filopodia. In addition, Ddvasp null cells have a defect in particle adhesion but internalize particles normally. Our results provide new insights into the function of DdVASP in controlling the actin cytoskeleton during chemotaxis and filopodia formation.