

EXAMINATION OF THE ROLE OF GROWTH FACTOR RECEPTORS AND THE INTEGRIN $\alpha 5\beta 1$ IN CD9 MEDIATED CELL MOTILITY

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Overexpression of tetraspanin CD9 in Chinese Hamster Ovary (CHO) cell expression model system promotes haptotactic cell migration to the extracellular matrix (ECM) protein fibronectin (FN). In the current report, we investigated the possible role of serum-containing growth factors and a CD9 molecular partner, integrin $\alpha 5\beta 1$, in CD9 promoted cell migration. CD9 expressing CHO cells (A6) retained their characteristic pro-migratory phenotype even after culturing in 1% serum media for 18 hrs, suggesting that CD9 promoted migration is due to CD9 influence on adhesion molecules rather than growth factor receptor activity. Integrin $\alpha 5\beta 1$ mediates adhesive functions of cells such as cell migration on FN. Altered expression or activity of $\alpha 5\beta 1$ dramatically affects the phenotypic properties of cells on FN. Since $\alpha 5\beta 1$ is expressed on CHO cells and serves as the major FN receptor, we investigated possible regulation of $\alpha 5\beta 1$ function by CD9 in promoting cell migration. Flow cytometry analysis revealed that cell surface density of integrin $\alpha 5\beta 1$ was not significantly altered because of CD9 over-expression. An anti- $\alpha 5\beta 1$ monoclonal antibody (PB1), earlier shown to inhibit the functions of $\alpha 5\beta 1$ on FN, selectively inhibited CD9 promoted cell migration. These data suggest that CD9 positively modulates $\alpha 5\beta 1$ interactions with the ECM ligand FN.

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