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RhoA Regulates E-Cadherin Intercellular Junctions in Oral Squamous Carcinoma Cells

Authors : Ga-Young Lee, Hyun-Man Kim

Abstract : The modulation of the cell-cell junction is critical in epithelial-mesenchymal transition during tumorigenesis. As RhoA activity is known to be up-regulated to dissociate cell-cell junction by contracting acto-myosin complex in various cancer cells, the present study investigated if RhoA activity was also associated with the disruption of the cell-cell junction of oral cancer cells. We studied SCC-25 cells which are established from oral squamous cell carcinoma if their E-cadherin junction (ECJ) was under control of RhoA. Interestingly, development of ECJ of SCC-25 cells depended on the amount of fibronectin (FN) coated on the culture dishes. Seeded cells promptly aggregated to develop ECJ on the substrates coated with a low amount of FN, whereas they were retarded in the development of ECJ on the substrates coated with a high amount of FN. However, it was an unexpected finding that total RhoA activity was lower in the dissociated cells on the substrates of high FN than in the aggregated cells on the substrates of low FN. Treating the dissociated cells on the substrates of high FN with LPA, a RhoA activator, promoted the development to ECJ. In contrast, treating the aggregated cells on the substrates of low FN with Clostridium botulinum C3, a toxin decreasing RhoA activity, dissociated cells concomitant with the disruption of ECJ. Genetical knockdown of RhoA expression by transfecting RhoA siRNA also down-regulated the development of ECJ in SCC-25 cells. Furthermore, PMA, an activator of protein kinase C (PKC), down-regulated the development of ECJ junction of SCC-25 cells on the substrates coated with low FN. In contrast, GO6976, a PKC inhibitor, up-regulated the development of ECJ of SCC-25 cells with the activation of RhoA on the substrates coated with high FN. In conclusion, in the present study, we demonstrated unexpected results that the activation of RhoA promotes the development of ECJ, whereas the inhibition of RhoA retards the development of ECJ in SCC-25 cells.

Keywords : E-cadherin junction, oral squamous cell carcinoma, PKC, RhoA, SCC-25
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