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Uterine epithelial cells require special phenotype plasticity for trophoblast adhesion

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Experiments on human uterine epithelial RL95-2 cells had previously demonstrated inversion of the asymmetrical apico-basal distribution of F-actin and of the small GTPase RhoA in response to binding of human trophoblastoid JAR spheroids. We now investigated the relationship between the degree of inversion of cytoplasmic architecture in uterine epithelial cells and preattachment/postattachment polarity. Therefore, we compared subcellular distribution of F-actin and RhoA along the apico-basal axis in RL95-2 cells with another type of human uterine epithelial cells, i.e. Ishikawa cells which are more polarized and less adhesive. Analysis was performed by confocal laser scanning microscopy before and after binding of JAR spheroids. Before contact to JAR cells, Ishikawa cells as well as RL95-2 cells showed significantly higher levels of F-actin and RhoA in the basal poles (*Ishikawa*: F-actin: 32 ± 8 grey scale values [gsv] apical vs. 51 ± 9 gsv basal, RhoA: 27 ± 6 gsv apical vs. 34 ± 6 gsv basal; *RL95-2*: F-actin: 28 ± 2 gsv apical vs. 41 ± 3 gsv basal, RhoA 24 ± 3 gsv apical vs. 37 ± 6 gsv basal). After contact, levels of F-actin and RhoA signals became equalized in the apical and basal regions of Ishikawa cells (F-actin: 103 ± 11 gsv apical vs. 106 ± 7 gsv basal, RhoA: 67 ± 9 gsv apical vs. 64 ± 7 gsv basal). In RL95-2 cells, however, contact with JAR spheroids led to an inversion of the original pattern (F-actin: 108 ± 17 gsv apical vs. 57 ± 7 gsv basal, RhoA: 55 ± 10 gsv apical vs. 25 ± 3 gsv basal). Thus, Ishikawa cells respond to JAR cell binding with a less extensive reorganization of cell architecture than RL95-2 cells. This may explain lower levels of adhesiveness for JAR spheroids in Ishikawa cells as in RL95-2 cells. Therefore, competence of phenotype modification may play an essential role in apical adhesiveness of uterine epithelium for trophoblast.