423 UTERINE EPITHELIAL CELL POLARITY AND ADHESIVENESS FOR TROPHOBLAST: IN-VITRO MODEL STUDIES USING CELL LINES.
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Embryo implantation involves initial formation of cell-to-cell contact between uterine epithelium and trophoblast. However, adhesive properties are normally not displayed by apical plasma membranes of epithelia. A recent hypothesis postulates that uterine epithelial cells (UECs) acquire adhesiveness of their apical cell pole for the implanting blastocyst during the receptive phase of cycle by partially down-regulating aspects of their epithelial phenotype.

In the present investigation, an in-vitro approach has been taken to study elements of UEC adhesiveness focusing on apico-basal cell polarity. We have examined human endometrial cell lines RL95-2 and HEC-1A, both of uterine epithelial origin. Adhesiveness of RL95-2 monolayers for choriocarcinoma cells (JAR spheroids) correlated with (a) random distribution of α6-, β1-, β4-integrin subunits over the entire cell membrane, (b) a perinuclear localization of microfilaments, intermediate filaments and microtubules, and (c) a lack of regular intercellular contacts. In contrast, non-adhesiveness of HEC-1A monolayers for JAR spheroids correlated with (a) basolateral distribution of integrins, (b) epithelium-like organization of cytoskeleton, and (c) regular intercellular contacts. As RL95-2 cells appeared to be adhesive, thereby lacking apico-basal cell polarity, we examined expression and distribution of the epithelial markers desmoplakin, E-cadherin, and ZO-1 which reportedly play a role in establishing apico-basal polarity. In RL95-2 cells desmoplakin and E-cadherin were randomly distributed over the entire cell membrane, and these cells did not express the tight junction-specific ZO-1 protein, in contrast to HEC-1A cells.

By extrapolation, these data on the two in-vitro cell lines are consistent with in-vivo observations suggesting that adhesiveness of UECs at receptivity might be due to temporary loss of certain features of cell polarity. Assembly of tight junctions and/or tight junction - cytoskeleton interactions might contribute to the regulation of these phenotypic alterations.