# Localization of uvomorulin, fodrin and actin in mouse embryos during compaction, decompaction-recompaction and blastulation

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Compaction, a critical event in the early mouse development, constitutes the beginning of trophoblast differentiation. Since compaction is attributed to the increasing in cell adhesion due to the cadherin uvomorulin and to the organization of cytoskeleton, and since uvomorulin is linked to the cytoskeleton, we examined by confocal microscopy the localization of uvomorulin, fodrin and actin in morulae during normal compaction, during various decompacting-recompacting assays and at the expanded blastocyst state. Decompacting treatments used are cytochalasin D and latrunculin (microfilament inhibitors), EGTA (calcium chelator), verapamil (calcium channel blocker), TFP (calmodulin inhibitor) and TMB-8 (calcium release inhibitor). The results indicate that uvomorulin deregionalizes when embryos are decompacted and that regionalization is recovered when recompaction is allowed; the same holds for fodrin, though to a lesser extent, while actin remains unchanged. Regionalization of uvomorulin