Gynecol Obstet 2018, Volume: 8
DOI: 10.4172/2161-0932-C1-025

Elimination of aneuploid cells in the early mammalian embryo

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Whole chromosomal aneuploidies - loss and/or gain of chromosomes from a euploid complement - are responsible for the low fecundity in humans. Here, we use mouse embryo as a model system to understand how mosaic aneuploidy affects embryonic development. To experimentally induce chromosome mis-segregation in the cleavage stage embryos, we inhibit the master spindle assembly checkpoint kinase Mps1, which results in a high percentage of aneuploid cells in the embryos. We introduce mosaicism in the system by making chimeras using euploid and aneuploid cells. Detailed analysis of such chimeras revealed that during blastocyst maturation abnormal cells contributing to the inner cell mass undergo preferential elimination up to 40%. Using an *in vitro* culture system capable of recapitulating *in vivo* mosaic embryos during peri-implantation development and time-lapse imaging, we have found that aneuploid cells were preferentially eliminated from the epiblast via apoptosis. Using pharmacological treatments to knockdown genes involved in autophagy, we have found the onset of autophagy-mediated apoptosis during embryogenesis. Overall, our findings suggest that p53-mediated autophagy promotes preferential apoptosis of aneuploid cells in early mouse mosaic embryos. This work gives an insight into the mechanisms behind sub-fertility, developmental defects and miscarriages during pregnancy.

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