A0312

In Vitro Development and Apoptosis in Haploid, Diploid Parthenotes and Fertilized Embryos

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Haploid parthenotes have been shown to be developmentally delayed compared with diploid parthenogenetic embryos in the mouse and pig. These developmental defects have been hypothesized to rusult from insufficient parthenogenetic activation, suboptimal *in vitro* culture conditions, or genemic imprinting. In the present study we compared the incidence of apoptosis and apoptosis related gene expression in pig haploid, diploid parthenotes and fertilized embryos. *In vitro* matured porcine oocytes were activated by electrical stimulation. Haploid activated oocytes with two polar bodies under stereomicroscopy were defined haploid parthenotes, oocytes with one polar body were defined as diploid parthenotes after 3h cycloheximide teatment. The morphological analysis of apoptosis in embryos was carried out using propidium iodide staining and terminal deoxynucleotidyl transferase mediated dUTP nick end labeling. The expression of Bcl-xL, Bak and P53 in haploid, diploid and *in vivo* fertilized blastocysts was determined using RT-PCR. Lower number of the haploid pig parthenotes developed to the morulae and blastocysts compared to the diploid parthnotes. Number of cells significantly lower in the haploid-derived blastocysts than diploid-derived it. Developmentally retarded haploid parthenotes exibited apoptosis at a significantly higher frequency than did diploid parthenotes and fertilized embryos. Level of Bcl-xL expression, diploid parthenotes similar to *in vivo*-derived it was higher than haploid parthenotes. However, Bak and P53 mRNA expression were not different among haploid, diploid, and fertilized embryos. This result suggested that parthenogenetic activation and parthenogenesis themselves do not cause apoptosis, but haploid increases the incidence of apoptosis in preimplantation embryos. Apoptosis may be due to decrease expression of Bcl-xL in haploid parthenotes developing *in vitro*.

Key words) ***Haploid, Genemic imprinting, Diploid, Apoptosis, RT-PCR***