Title: L-Carnitine improves blastocyst developement rate and reduces DNA damage in mouse embryos.
Objective: Cytoplasmic fragmentation may be present in the in-vitro cultured embryos. This fragmentation may arise from apoptosis. Apoptosis is associated with changes in the mitochondrial membrane potential. Recent studies show that ooplasmic injection of isolated mitochondria can rescue oocyte fragmentation in mice. L-Carnitine (LC) is able to stabilize the mitochondrial membranes potential, increase the supply of energy and protect the cell from apoptotic death. In a pilot study we have shown that L Carnitine can improve the blastocysts development rate (%BDR) in 2-cell embryos at low concentrations (0.3mg/ml). However, the BDR rates decreased at higher concentrations (>0.6mg/mL). Our objective was to examine the level of apoptosis in blastocysts at concentrations of LC that were not embryotoxic.

Design: Prospective in vitro study.

Materials and Methods: A total of 60 two-cell mouse embryos were randomly divided into 3 groups: group 1: control (n = 20; HTF media only); groups 2: LC 0.3 mg/mL (n = 20) and group 3: LC 0.6mg/mL (n = 20). Embryos were incubated at 37°C in 5%CO₂ for 72h. Assessment of embryos development was done by examining the %BDR. After TUNEL staining, the blastocysts were examined by confocal microscopy. Apoptosis was measured by examining the percentage of TUNEL positive blastomeres.

Results: Comparison of %BDR and apoptosis between control and 0.3 and 0.6mg/mL of L-Carnitine.

Conclusions: LC is not embryotoxic at low concentrations and may even improve the blastocyst development rate. Level of apoptosis is not altered by incubating mouse embryos with LC. Supplementation of embryo culture media with LC may offer a novel approach to improve the embryogenesis in IVF.

Support: None