46. EFFECT OF GM-CSF ON THE IN VITRO DEVELOPMENT OF PORCINE EMBRYOS

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Granulocyte-macrophage colony-stimulating factor (GM-CSF) is expressed in the female reproductive tract during early pregnancy and has been implicated in the regulation of preimplantation embryo development in several species. Culture of human 2- to 4-cell embryos in medium supplemented with recombinant human GM-CSF increased the number of blastocyst cells allocated to the inner cell mass (ICM) and reduced the number of apoptotic cells after 5 days (1). In this study we assessed the effect of recombinant porcine GM-CSF (rpGM-CSF) on the development of porcine embryos. One- and 2-cell embryos (n = 274) were surgically collected as described previously (2) from 18 Large White × Landrace gilts. In 5 replicates of the experiment, embryos from each donor were allocated across treatments. Serum-free culture medium of Tn5 cells infected with the AcPGM virus was the source of rpGM-CSF. The biological activities of rpGM-CSF in AcPGM-infected cell culture supernatants was previously demonstrated by porcine bone marrow cell proliferation and haematopoietic cell colony formation assays (3). Embryos were cultured for 7 days at 38.5°C in either North Carolina State University 23 (NCSU23) medium alone, or NCSU23 medium supplemented with supernatants to give a final GM-CSF concentration of 0, 1 or 10 ng/mL. The proportion of embryos developing to the hatching and hatched blastocyst stages was increased by the addition of supernatants containing 0, 1 and 10 ng/ml GM-CSF (62%, 59% and 59%, respectively) compared with NCSU23 medium alone (31%). Addition of 1 ng/mL GM-CSF increased the number of blastocyst cells allocated to the ICM (15.5 ± 1.9 cells) compared with 0 and 10 ng/ml GM-CSF (11.1 ± 1.7 and 8.2 ± 0.9 cells, respectively). The results indicate that factor(s) present in the supernatants other than GM-CSF affected blastocyst formation. Despite this, GM-CSF was found to influence the allocation of cells within the developing porcine embryo.

(1) Sjöblom *et al*. (2002) *Biol. Reprod.* **67**: 1817–1823. (2) Nagashima *et al*. (1994) *Biol. Reprod*. **51**: 618–622. (3) Inumaru *et al*. (1998) *Immunol. Cell Biol.* **76**: 195–201.

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