Animal Reproduction

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EMBRYOLOGY, DEVELOPMENTAL BIOLOGY, AND PHYSIOLOGY OF REPRODUCTION

Oleic acid supplementation affects prostaglandin synthesis in bovine trophoblast cells cultured *in vitro*

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In cattle early embryonic mortality, caused by failures in maternal recognition of pregnancy (MRP), is one of the major causes of reproductive failures. Oleic acid (OA) determines modifications in the synthesis of prostaglandins that may favor MRP. The objective was to determine the effects of supplementation with OA (Sigma, O1383) in the *in vitro* culture of bovine trophoblastic cells (CT-1) on the synthesis of prostaglandin E2 (PGE2) and prostaglandin F2α(PGF2α). The CT-1 were cultured in plates with culture medium supplemented with 10% fetal bovine serum (FBS), incubated at 38.5°C in a humidified atmosphere with 5% CO2. After 5 days of cultivation, they received the same medium free of FBS for 24 hours. Then, the wells received 4 mL of medium without FBS containing AO at concentrations of 0, 50, 100, 200, 500 or 1000 µM; for 48 or 72 hours. For each concentration and time, a well was constituted in each repetition, with a total of 5 repetitions. 300 µL of medium was collected from each well 48 or 72 hours after administering the treatments. PGE2 and PGF2α concentrations were determined by enzyme- linked immunosorbent assay. Statistical analysis was performed using PROC MIXED from SAS. The concentration of PGF2α at 48 hours was higher ($P < 0.0001$) in treatments with 50 µM, 100 µM, 200 µM and 500 µM of OA (88.97 ± 7.11; 95.31 ± 7.11; 114.88 ± 7.11 and 107.83 ± 7.11 ng/ml; respectively) compared to the control group (61.52 ± 7.11 ng/ml), the 1000 µM OA treatment (58.44 ± 7.11 ng/ml) did not differ from the control group. The concentration of PGF2α at 72 hours was higher ($P < 0.0001$) in treatments with 50 µM, 100 µM, 200 µM and 500 µM of OA (64.08 ± 2.89; 76.45 ± 2.89; 70.31 ± 2.89 and 59.31 ± 2.89 ng/ml; respectively) compared to the control group (49.40 ± 2.89 ng/ml), the 1000 µM OA treatment (39.41 ± 2.89 ng/ml) was lower than in the control group. CT-1 supplemented with 200 µM, 500 µM and 1000 µM of OA showed an increase ($P = 0.0151$) in PGE2 synthesis within 48 hours (20.61 ± 3.39; 20.11 ± 3.39 and 19.11 ± 3.39 ng/ml, respectively) compared to the control group (4.80 ± 2.93 ng/ml). At 72 hours, there was a tendency ($P = 0.0872$), towards an increase in PGE2 synthesis, when CT-1 supplemented with 50 µM, 200 µM and 1000 µM of OA (18.60 ± 3.60; 20.20 ± 3.60 and 19.00 ± 3.60 ng/ml; respectively) were compared to the control group (5.40 ± 3.60 ng/ml). In groups treated with OA for 48 hours, the PGE2/PGF2α ratio was lower ($P = 0.0460$) when CT-1 was supplemented with 200 µM and 500 µM of OA (0.62 ± 0.06 and 0.56 ± 0.06 ng/ml) compared to the control group (0.82 ± 0.06 ng/ml). At 72 hours, there was a tendency to decrease the PGE2/PGF2α ratio ($P = 0.0834$) only when the TC-1 were supplemented with 500 µM of OA (0.62 ± 0.10 ng/ml) compared to the control group (0.93 ± 0.09 ng/ml). In conclusion, OA supplementation at a concentration of 1000 µM decreases PGF2α synthesis with 72 hours and increases PGE2 synthesis with 48 hours in the *in vitro* culture of CT-1.