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Effect of Elevated Temperature on Gene Expression of Bovine Embryos

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Low viability of mammal embryos is found when the animals are exposed to high temperature, reducing reproductive efficiency and, consequently, productivity. This scenario can get worse with the global warming, mainly for livestock species living in the tropics. We aimed to evaluate the effect of heat stress on expression of some genes in fertilized or parthenogenetic bovine embryos in order to highlight the mechanisms involved in embryo adaptation to elevated temperatures. Oocytes were in vitro fertilized or activated with ionomycin to induce parthenogenesis. Embryos at 44h post-fertilization or parthenogenesis were cultured under 41°C for 12h to promote the heat stress or under isothermal conditions (38.5°C) to constitute the non-stressed groups (control). The embryos were thereafter cultured at 38.5°C for 124h until blastocyst stage. Messenger RNA isolated from fertilized or parthenogenetic blastocysts was transcribed to cDNA and amplified by real-time PCR. The expression of heat shock 70kDa protein 1A (HSP70.1), peroxiredoxin 1 (PRDX1), glucose transporter type 1 (GLUT1), glucose transporter type 5 (GLUT5) and insulin-like growth factor 1 receptor (IGF1R) genes were analyzed and compared between stressed and non-stressed fertilized or parthenogenetic embryos using the pair-wise, fixed reallocation randomization test (REST software). Expression of glyceraldehyde-3-phosphate dehydrogenase and beta-actin genes were used as calibrator. In vitro fertilized embryos that underwent heat stress showed higher ($P<0.05$) level of PRDX1 transcripts than the non-stressed embryos. Heat-stressed parthenogenetic embryos showed lower ($P<0.05$) level of GLUT1 and GLUT5 transcripts than the non-stressed embryos. No difference was found for the other genes. The PRDX1 gene encodes a protein involved on anti-oxidative response whereas the GLUT1 and GLUT5 genes encode proteins involved on energy substrate uptake. Switch on their expression may be an embryo attempt to keep its viability and suggests that, regardless fertilization or parthenogenesis, epigenetic modifications may be involved on mammal embryo adaptation to elevated temperature.

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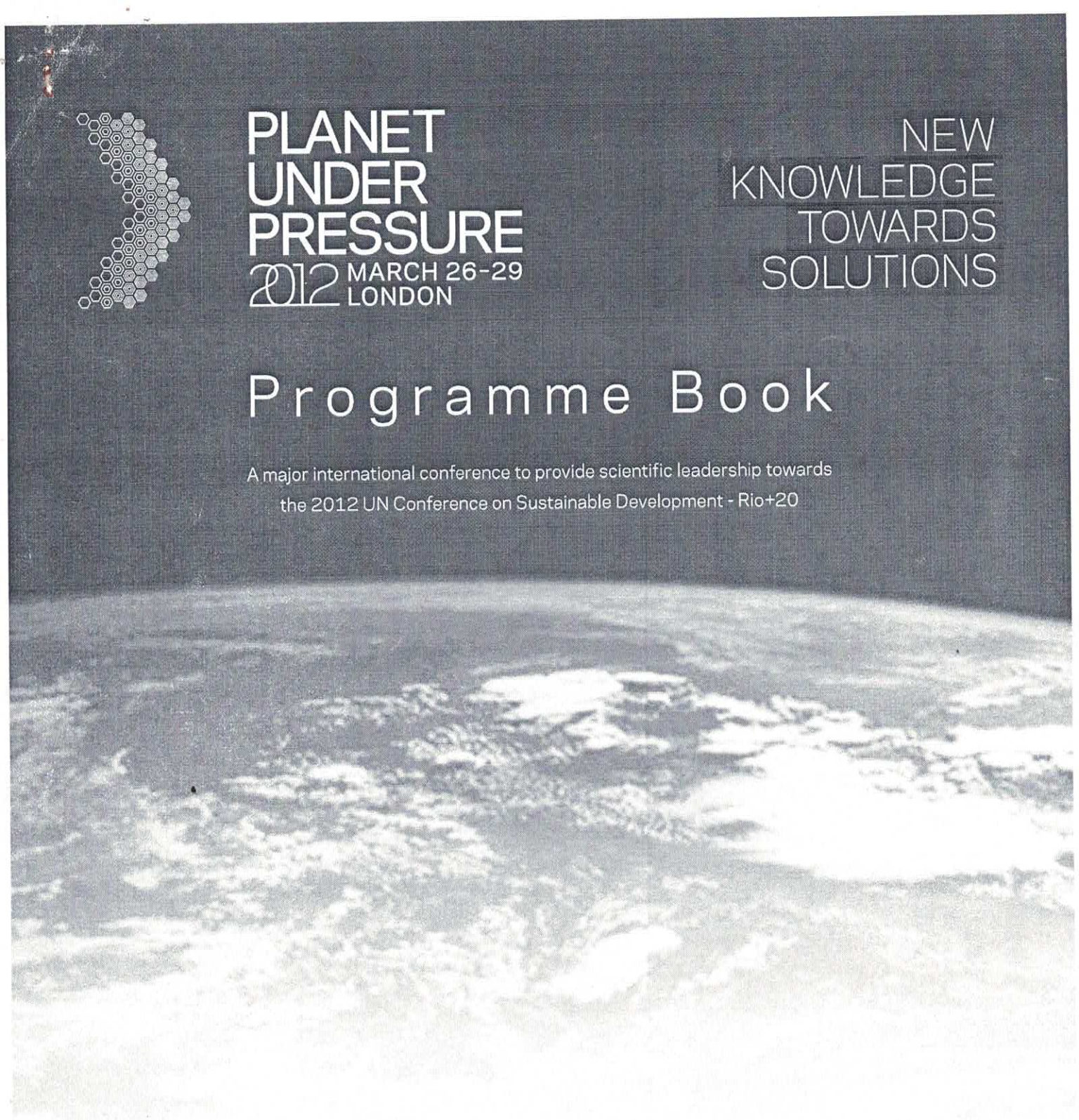

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