Deoxynivalenol induces the endoplasmic reticulum stress-mediated apoptosis via the IRE1/JNK/CHOP signal pathway in porcine embryos

Hyo-Jin Park[1,2]; Ye-Won Kim[1,2]; Seul-Gi Yang[2,3] and Deog-Bon Koo[1,2,3]

1. Department of Biotechnology, Daegu University, 201 Daegudae-ro, Jillyang, Gyeongsan, Gyeongbuk 38453, Republic of Korea

2. DU Center for Infertility, Daegu University, 201 Daegudae-ro, Jillyang, Gyeongsan, Gyeongbuk 38453, Republic of Korea

3. Department of Companion Animal Industry, Daegu University, 201 Daegudae-ro, Jillyang, Gyeongsan 38453, Republic of Korea

The cytotoxic mycotoxin deoxynivalenol (DON) has been reported to adversely affect oocyte maturation and embryo development in pigs. The interplay between cell apoptosis and endoplasmic reticulum (ER) stress has been increasingly recognized as important in embryogenesis. However, the role of the inositol-requiring enzyme 1 (IRE1)/c-Jun N-terminal kinase (JNK)/C/EBP-homologous protein (CHOP) pathway within the unfolded protein response (UPR) signaling in DON-induced apoptosis of porcine embryos remains unclear. In our study, we found that exposure to DON (0.25 μM) led to a significant decrease in cell viability up to the blastocyst stage. This was associated with the onset of cell apoptosis via the IRE1/JNK/CHOP pathways, as a response to ER stress in porcine embryos. Also, our validation via quantitative PCR (qPCR) results confirmed the upregulation of UPR signal related transcription factors in DON-induced porcine blastocysts. And IRE1/JNK/CHOP signal activations by Western blot analysis, indicating the instigation of ER stress-associated apoptosis by DON exposed embryos in pigs. urther experiments have shown that taurourso deoxycholic acid (TUDCA) mitigates the ER-stress induced by DON in porcine embry os, indicating that TUDCA can counteract the toxicity of DON on early embryonic d evelopmental competence in pigs during the IVC process. In conclusion, DON exposure impairs the embryonic developmental ability by ER-stress mediated apoptosis via IRE1/JNK/CHOP signal in pigs. These results suggest that they can also be a cause of decreased fertility and embryonic development in human.

Keywords: Deoxynivalenol, Endoplasmic reticulum stress, Unfolded protein response, Tauroursodeoxycholic acid, Porcine embryo

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