Does Lipopolysaccharide Influence Bovine Endometrial Response to Interferon Tau?

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The uterine environment during the peri-implantation period is crucial for conceptus growth, elongation and attachment leading to a successful pregnancy establishment in cattle. Postpartum uterine disease is a major factor affecting fertility in high-producing dairy cows through disruption of ovarian and uterine function. Lipopolysaccharide (LPS), a well-known endotoxin and the major component of the outer membrane of Gram-negative bacteria, causes poor uterine receptivity by inducing excessive inflammation at the maternal-fetal interface. We aimed to investigate the effect of interferon tau (IFNT), the conceptus-derived maternal recognition signal, on bovine endometrial gene expression in the absence or presence of LPS. Endometrial explants were collected at a local abattoir from Holstein Friesian cows (n=4) during the mid-luteal stage of the oestrous cycle, and cultured in RPMI medium supplemented with 5% fetal calf serum without (control), or with IFNT (100 ng/mL), LPS (1 μ g/mL), or both IFNT and LPS for 24 h in 5% CO₂ in humidified air. Gene expression was analysed by RT-qPCR. Treatment effects were considered significant at P<0.05. Incubation with IFNT upregulated (P<0.05) the well-known classical interferon-stimulated genes (ISGs: ISG15, OAS1, MX1 and MX2) as well as selected ISGs (CMPK2, IFI35, TRIM38, TNFSF10) from our previous study (Talukder et al., 2023 doi: 10.1016/j.theriogenology.2023.07.033) and downregulated expression of *IL1B* in endometrial explants. Incubation with LPS increased (P<0.05) expression of inflammation-related genes (TNFA, IL6, and IL1B) as well as ISG15 and MX1 in endometrial explants but did not alter endometrial response to IFNT in terms of the studied ISGs. These results suggest that the expression of ISGs, upregulated by conceptus-derived IFNT, is not altered in the endometrium in the presence of LPS; however, the increased expression of inflammation-related genes induced by LPS indicate an altered endometrial immune response that may be associated with compromised pregnancy establishment.

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