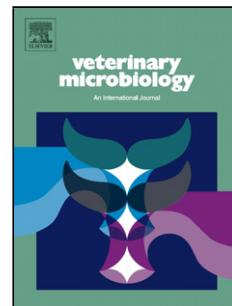


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Authors: Sandra Genís, Àlex Bach, Anna Arís



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Effects of intravaginal lactic acid bacteria on bovine endometrium: implications in uterine health

Sandra Genís¹, Àlex Bach^{1,2}, Anna Arís^{1,*}

¹Department of Ruminant Production, IRTA (Institut de Recerca i Tecnologies Agroalimentàries). 08140, Caldes de Montbui, Spain.

²ICREA (Institució Catalana de Recerca i Estudis Avançats), 08010, Barcelona, Spain.

*Corresponding author. Tel.: +34934674040. E-mail address: anna.aris@irta.cat

Highlights

- LAB treatment applied intra-vaginally does not reach the endometrium.
- There is no modulation of inflammatory genes.
- LAB decreases the amount of *E. coli* in the endometrium.
- LAB decreases expression of infection markers *B-defensins* and *MUC1*.

Abstract

Infection and inflammation of the endometrium after calving compromise uterine health, contributing to decreased reproductive efficiency in dairy cows. Twenty multiparous cows were distributed in two groups and treated intra-vaginally with a combination of lactic acid bacteria (LAB) composed by *Lactobacillus rhamnosus*, *Pediococcus acidilactici*, and *Lactobacillus reuteri*, or with a sterile carrier (CON) twice per week during 3 wk. At the slaughterhouse, vaginal and endometrial swabs were taken for *E. coli* and *Lactobacillus* quantification. Endometria were collected and cut forming explants that were analyzed for the expression of 10 genes related to innate immunity and infection or submitted to an *ex vivo* inflammation model. In the *ex vivo* experiment, explants were infected with *E. coli* or inflamed by treating them with IL-1 β and also *E. coli*. The secretion of IL-8, IL-1 β , and IL-6 was evaluated by ELISA in the supernatants of the *ex vivo* cultures. *Lactobacillus* counts did not differ between endometria of LAB and CON cows, although *E. coli* vaginal counts tended to be lower in LAB than in CON cows. The expression of *B-defensins* and *MUC1*, indicators of infected uterus, was down-regulated in explants of LAB-treated cows. No differences were observed between LAB and CON explants in the *ex vivo* inflammation

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