

Thyroid hormones and cortisol in lactating cows after intramammary LPS or *Escherichia coli* administration

L. Senepart^{1*}, K. Winters¹, A. Gubbiotti², S. Lefever¹, R.M. Bruckmaier³, J.W. Blum³, G. Berton², E. Trevis², C. Burvenich¹.

¹ Milk Secretion and Mastitis Research Center (MMRC), Faculty of Veterinary Medicine, Ghent University, Belgium

² Inst. of Zootechnics, Fac. of Agriculture, Università Cattolica S. Cuore, Piacenza, Italy

³ Division of Nutritional Pathology, Faculty of Veterinary Medicine, University of Berne, Switzerland

*Corresponding Author: Lukas.Senepart@UGent.be

Introduction

The incidence of coliform mastitis is highest around parturition and during early lactation³. During growth and destruction of *E. coli*, lipopolysaccharides (LPS) are released in great amounts. Administration of LPS and *E. coli* mastitis share the same clinical signs and both mediate the release of inflammatory cytokines⁴. In this study, we compared the effects on endocrine changes using two strategies (i.e. challenge with *E. coli* and LPS) to induce mastitis.

Materials and Methods

Twelve healthy East Flemish Red Pied cows were accepted in the study. Six cows were infected with 1×10^4 cfu/quarter of *E. coli* strain P4:O32 (A.Hill, Compton, U.K.) while the other six cows were infused with 500 µg/quarter of endotoxin (LPS) from *E. coli* strain O111:B4, in the left front and rear quarters. The cows were transferred in individual tie-stalls one week before the challenge. They were fed with approximately 8 kg of concentrates, and were allowed to eat hay and drink water *ad libitum*. Blood samples were collected daily, 1 hour after morning milking (i.e. 1.5 hours after the morning meal) on day -1, +1, +2, +3, +6 and +9. On day 0, blood samples were collected immediately before infection (0 hours) and every 2 hours from 2 up to 14 hours after inoculation. Plasma levels of cortisol were analysed using the methods of Blum *et al.*²; T4, T3 and rT3 were analysed using the methods described by Bertoni *et al.*¹. Data were analyzed with a two way ANOVA, including group and time in the model, using the statistical analysis program package Statistix (Analytical Software, Tallahassee, FL, USA).

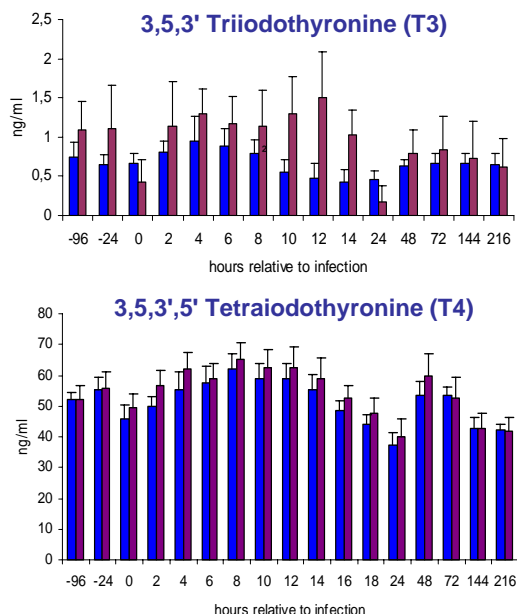


Figure 1: Mean changes in T3 and T4 in bloodplasma of healthy cows after i.mam. *E. coli* LPS (500 µg/quarter; blue areas) or i.m. infection with *E. coli* (1×10^4 cfu/quarter; purple areas).

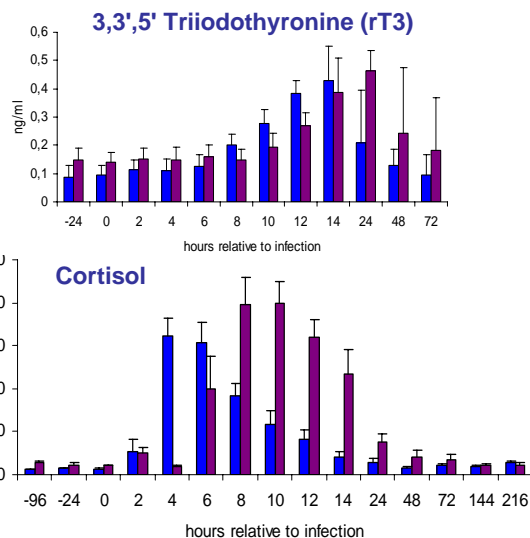


Figure 2: Mean changes in rT3 and cortisol in bloodplasma of healthy cows after i.mam. *E. coli* LPS (500 µg/quarter; blue areas) or i.m. infection with *E. coli* (1×10^4 cfu/quarter; purple areas).

Results

- T3 shows an increase in both groups and this increase was more pronounced after *E. coli* infection then after LPS infusion. 24 hours post infection however, an all time low in T3 (and T4) is seen.
- The rT3 profile shows a clear rise in both groups. The rise in rT3 occurs later in the *E. coli* group.
- Plasma cortisol levels increases in a spectacular way in both groups. The increase occurs later and is more pronounced in the *E. coli* group compared to the LPS group.

Discussion

Intramammary infusion of LPS or *E. coli* infection induced a release of cortisol and rT3 in blood plasma of cows. The decrease in T3 and T4 seen around 18–24 hours post inoculation seems to be correlated with the increase of rT3 in the same period. This may be a result of the conversion of T3 and T4 to rT3 in the peripheral tissues. This inactivation of thyroid hormones may be a countermeasure to avoid hyperthermia after disease. Cortisol may have role in the conversion of T3 and T4 to rT3 which would explain its rise⁵. The changes in concentration of the parameters were induced faster after i.mam. LPS then compared with *E. coli* administration. LPS can directly activate macrophages and/or epithelium in the mammary gland to release inflammatory cytokines through binding to the CD-14 receptor⁴. During *E. coli* mastitis, LPS first has to be released by bacteria before cells can be activated, so the onset of the changes occurs later. However, its effects appeared more pronounced than LPS infusion because cortisol remained higher for a longer time.

References

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