

# **Pathogen specific response of the bovine mammary gland to lipopolysaccharide from *E. coli* and lipoteichoic acid from *S. aureus***

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# Introduction

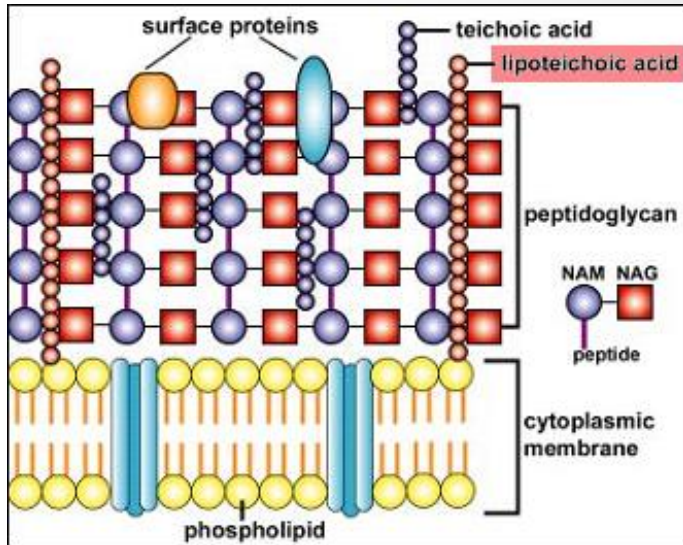
- Mastitis – common disease, economic losses
- Pathogens
- E. coli and S. aureus
- gram-positive, gram-negative
- Endotoxins

## Aim of the study

Immune response of the bovine mammary gland towards cell wall components of different pathogens

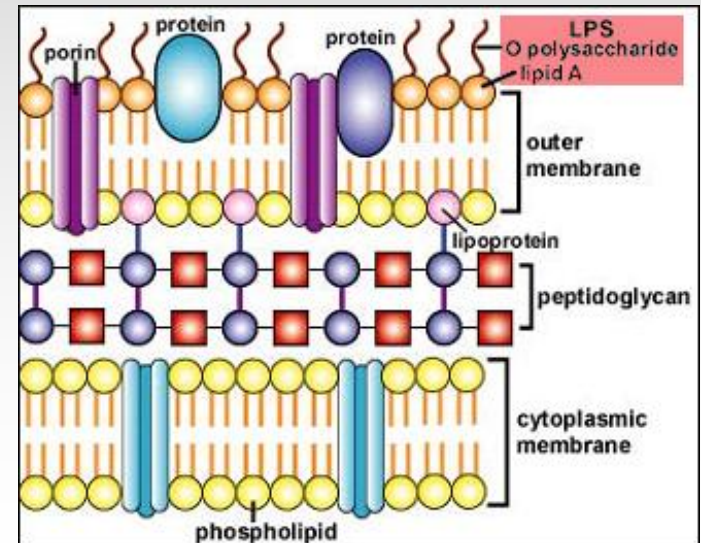
- LTA from *S. aureus*
- LPS from *E. coli*

## LTA vs. LPS



- LTA (Lipoteichoic acid):
  - in Gram-positive bacteria like *S. aureus*
  - component of the capsule (murein capsule)
  - *S. aureus* causes subclinical mastitis

- LPS (Lipopolysaccharide):
  - in Gram-negative bacteria like *E. coli*
  - component of the outer membrane
  - *E. coli* causes mainly clinical mastitis



## Experimental design

Challenge  
with LPS  
or LTA



Mammary tissue



Sampling at 0, 6, and 12 h

Milk

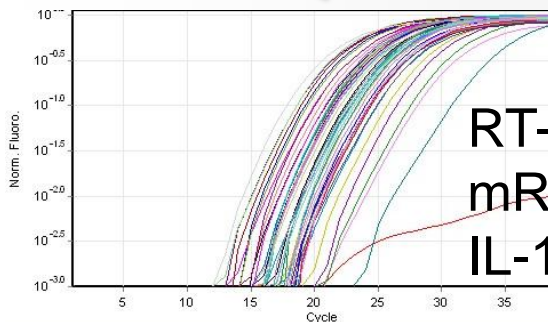


SCC



TNF $\alpha$  by RIA

LDH by  
enzymatic test



RT-qPCR:  
mRNA of TNF $\alpha$ ,  
IL-1 $\beta$ , IL-8, Lf

## Parameters

### **Milk cells: SCC (Somatic Cell Count)**

- first line of defense in the udder
- migration into the udder during infection

### **TNF $\alpha$ : Tumor Necrosis Factor $\alpha$**

- pro-inflammatory cytokine
- recruitment of leukocytes
- stimulating the production of secondary mediators

### **LDH: Lactate Dehydrogenase**

- secreted during an inflammatory process by leukocytes  
and damaged cells of the udder's epithelial and interstitial cells



# Immune Factors

## **IL-1 $\beta$ : Interleukin-1 $\beta$**

- pro-inflammatory cytokine
- many effects similar to those of TNF $\alpha$
- mediates local and systemic inflammatory response

## **IL-8: Interleukin-8**

- chemokine
- recruits neutrophils
- induced by exogenous and endogenous pro-inflammatory stimuli

## **Lf: Lactoferrin**

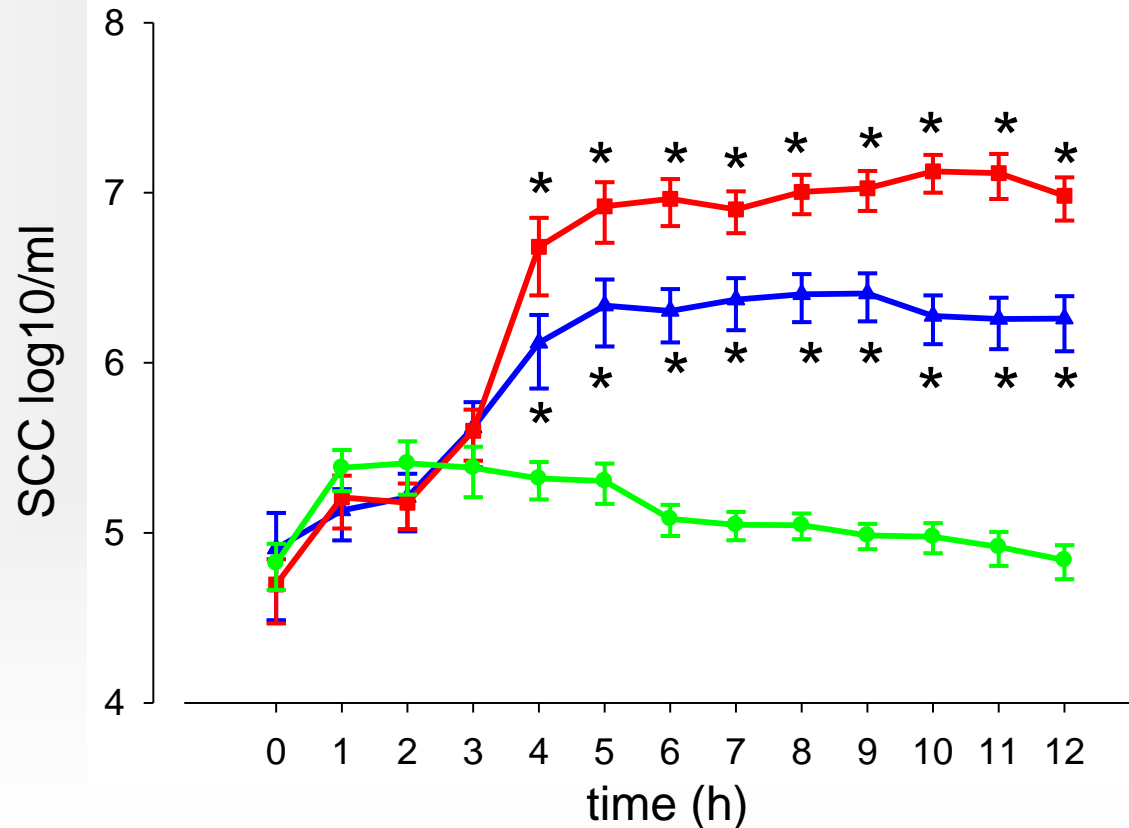
- bactericidal glycoprotein with various properties
- secreted by mammary epithelial cells and milk cells

# Results



## Milk: Somatic Cell Count

- ▲ LTA 10 $\mu$ g (n=8)
- LPS 0.2 $\mu$ g (n=8)
- Control (n=16)

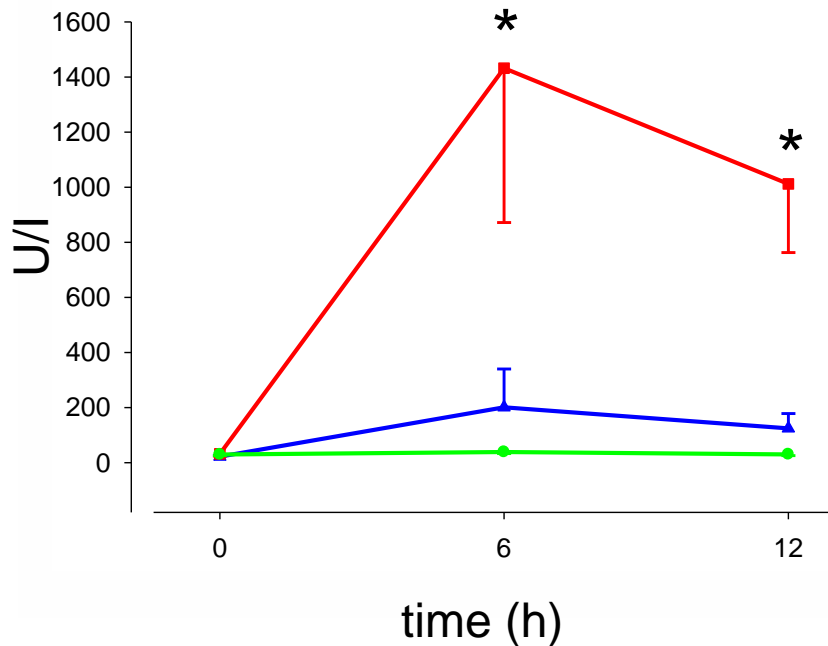


\* Means differ significantly ( $p \leq 0.05$ ) to time 0 h

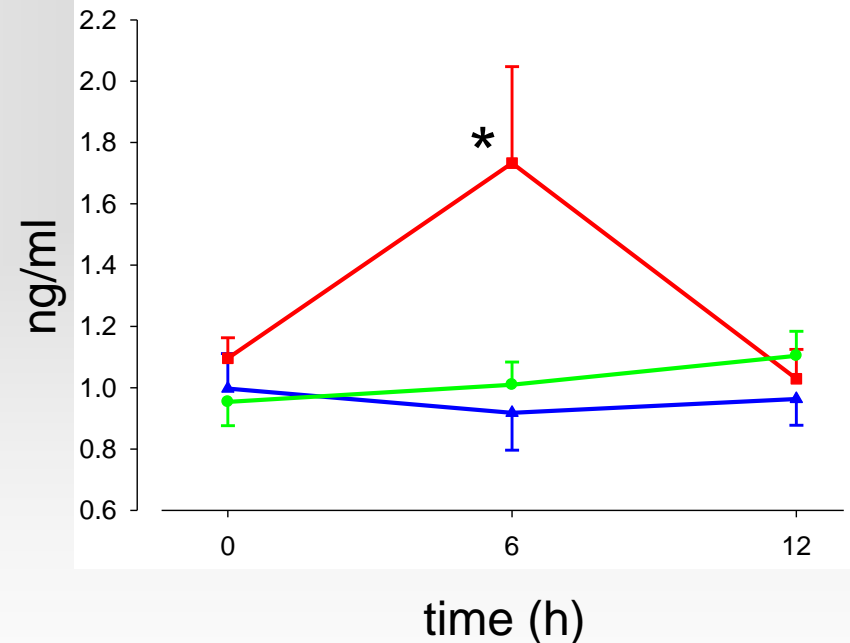
## Milk: TNF $\alpha$ and LDH

- ▲ LTA 10 $\mu$ g (n=8)
- LPS 0.2 $\mu$ g (n=8)
- Control (n=16)

### LDH



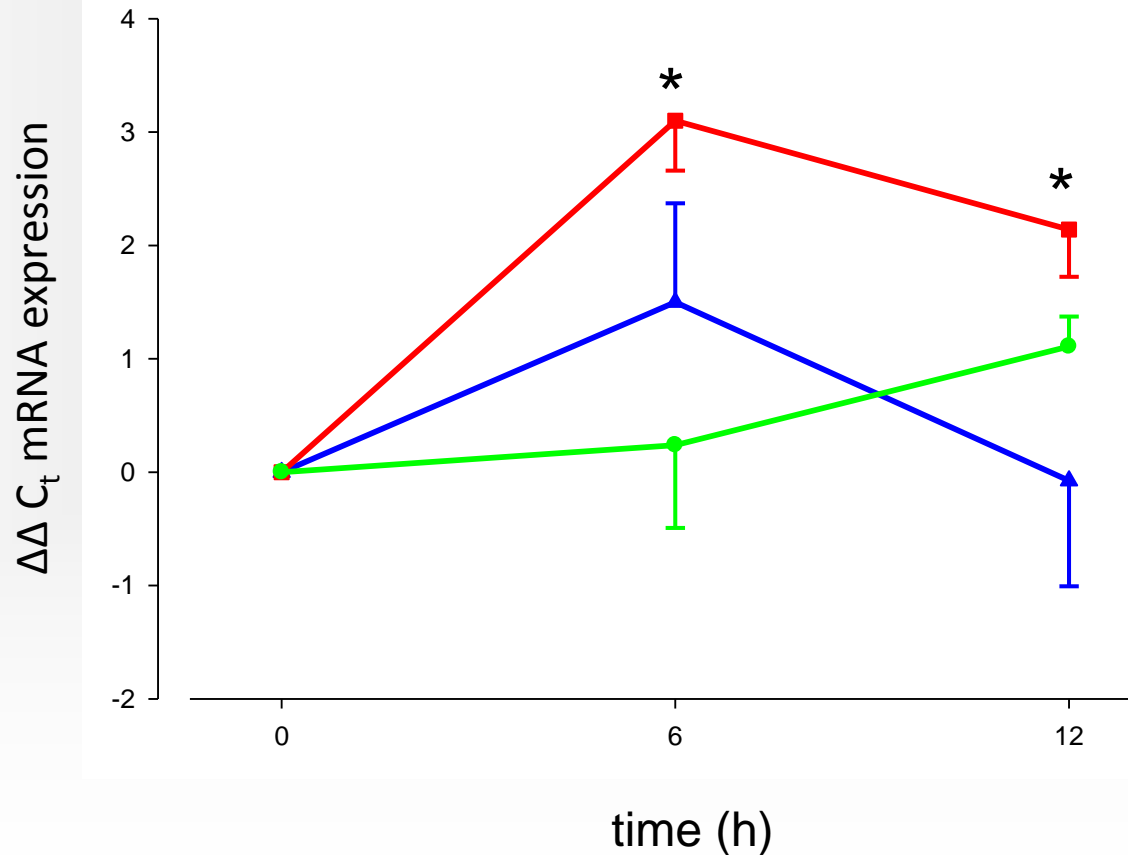
### TNF $\alpha$



\* Means differ significantly ( $p \leq 0.05$ ) to time 0 h

## Mammary tissue: $\text{TNF}\alpha$

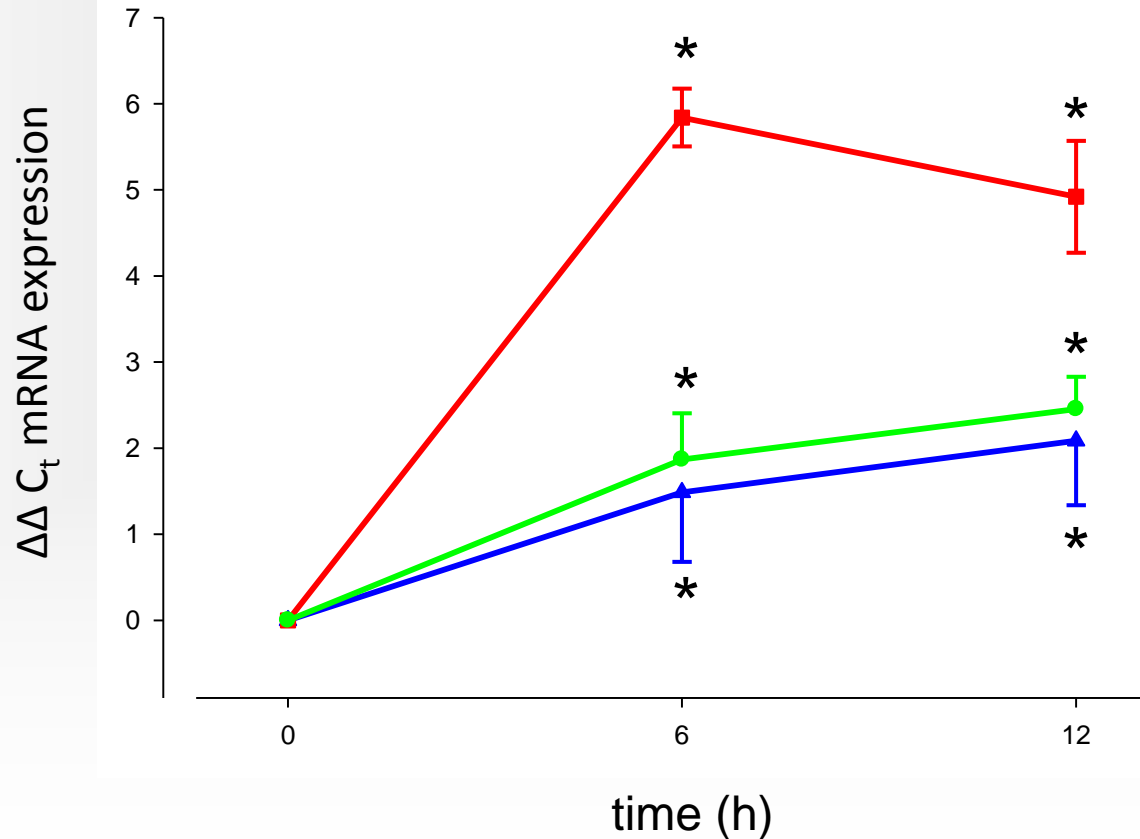
- ▲ LTA 10 $\mu\text{g}$  (n=8)
- LPS 10 $\mu\text{g}$  (n=5)
- Control (n=13)



\* Means differ significantly ( $p \leq 0.05$ ) to time 0 h

## Mammary tissue: IL-1 $\beta$

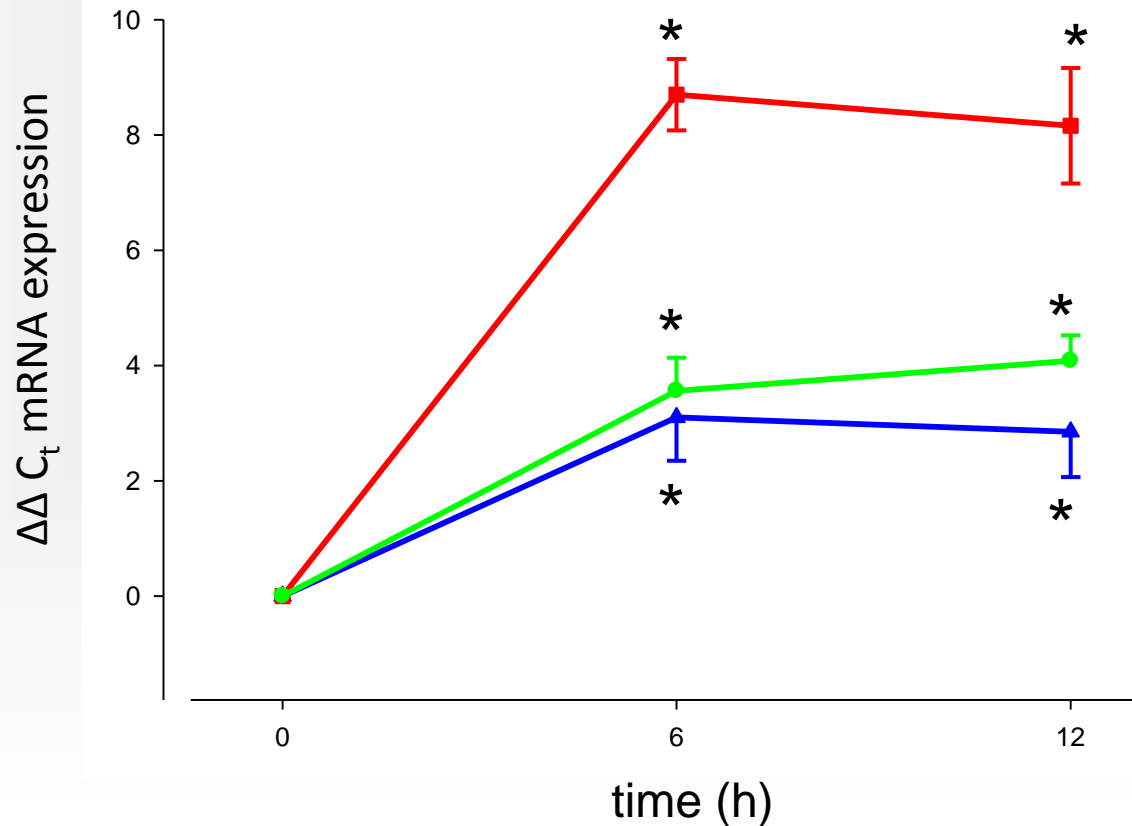
- ▲ LTA 10 $\mu$ g (n=8)
- LPS 10 $\mu$ g (n=5)
- Control (n=13)



\* Means differ significantly ( $p \leq 0.05$ ) to time 0 h

## Mammary tissue: IL-8

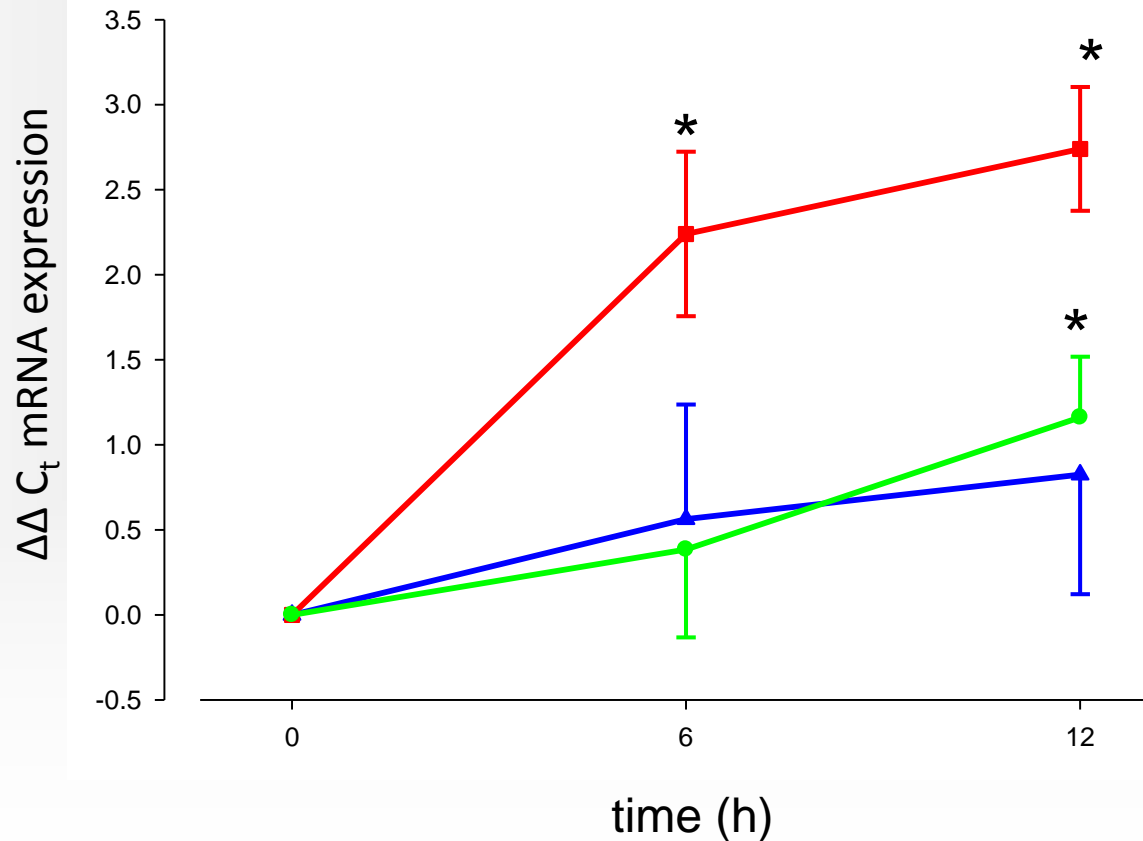
- ▲ LTA 10 $\mu$ g (n=8)
- LPS 10 $\mu$ g (n=5)
- Control (n=13)



\* Means differ significantly ( $p \leq 0.05$ ) to time 0 h

## Mammary tissue: Lactoferrin

- ▲ LTA 10 $\mu$ g (n=8)
- LPS 10 $\mu$ g (n=5)
- Control (n=13)



\* Means differ significantly ( $p \leq 0.05$ ) to time 0 h

## Summary and Conclusion

- LPS induced a stronger response of the measured factors than LTA despite similar SCC response.
- The present results are consistent with earlier investigations which showed a reduced and slower reaction of TNF $\alpha$  and IL-8 towards bacterial infection with *S. aureus* than with *E. coli*



Thanks for your attention!



Questions?