

Author's feedback against Editor's comments:

Different pathogens, such as *Escherichia coli* and *Staphylococcus aureus*, can be responsible for different outcomes of mastitis. Usually, to determine if intramammary challenge with the endotoxins lipopolysaccharide (LPS), from *E. coli*, and lipoteichoic acid (LTA), from *Staph. aureus*, induce different immune responses in cells and mammary tissue. LPS induces *E. coli* (G-) receptor TLR4, whereas LTA (G+) receptor for TLR2.

1. In this article aim was to assay the expression of an important TLR2 gene in naturally infected cows with sub-clinical and clinical mastitis caused by *E. coli* (G-). It seems against the principle LPS from *E. coli* vs TLR2 expression.

Though Lipoteichoic acid is a major constituent of cell wall of gram-positive bacteria, some of the research article has also mentioned that the Lipoteichoic acid is constituent of cell wall of gram negative bacteria (Included reference in the revised version).

2. In addition, as you point out in ref 8: CpG-DNA also promoted the expression of its specific receptor TLR-9 mRNA in mammary tissue. Not *E. coli* to TLR2.

By mistake we have included a wrong reference (Ref No.8). Now, we are corrected and included in the revised manuscript.

Authors can demonstrate TLR4 data rather than this unusual TLR2.

We carefully considered the reviewers comments, and tried to do our best to respond to the points raised and have made necessary changes accordingly to their indications in the manuscript.