Characterization of gene expression profiles to chronic infection with Mycobacterium avium subspecies paratuberculosis

Melvang, Heidi Mikkelsen; Grønbæk, Betina Chemnitz; Brogaard, Louise; Jakobsen, Jeanne Toft; Jungersen, Gregers

Publication date:
2015

Document Version
Publisher's PDF, also known as Version of record

Link back to DTU Orbit

Citation (APA):

General rights
Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.
Mycobacterium avium subsp. paratuberculosis (Map) causes paratuberculosis, a chronic enteritis of ruminants. The aim of the study was to use high-throughput reverse transcriptase (RT) qPCR to describe intestinal gene expression patterns in response to different levels of Map infection with a large panel of immunologically relevant genes.

For the study we selected samples of 6 calves that were all experimentally infected with Map at two weeks of age and based on serology, histology and Map tissue load were classified as protected (n=2) or unprotected (n=2) after vaccination, or un-vaccinated infected controls (n=2). From each calf, 7 intestinal tissue samples and 3 lymph node samples, collected at 10 months of age, were used for cDNA synthesis. Expression of a total of 37 selected genes including inflammatory, Th1 and Th17 related genes were explored.

The results showed that Map infection, as expected, leads to increased expression of local IFN-γ. Expression of IL-10 also increased as a result of Map infection, and this increase was more correlated to the amount of Map than IFN-γ, indicating a shift towards a regulatory environment as infection progress. Th17-mediated immune responses were suppressed at this stage. Gene expression of all other genes could not be interpreted in relation to infection status.

High throughput RT qPCR can be used for exploring gene expression patterns in response to Map infection but larger study groups are needed to fully understand which are key mechanisms and pathways responsible for protection or disease.