Galectin-1 modulates mucosal immune response against *Yersinia enterocolitica* infection

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**Introduction**

The mucosa of the gastrointestinal tract is continuously exposed to a myriad of antigens and microorganisms, however, only a limited number of which enter the body and cause disease. The gut-associated lymphoid tissue (GALT) comprise organized tissues such as the Peyer’s patches (PP) and mesenteric lymph nodes (MLN) that are generally considered to be inductive sites of the intestinal mucosae immune system.

Galectin-1 (Gal-1), an evolutionarily conserved β-galactoside-binding protein, has key roles in a variety of physiologic and pathologic processes. These functions include suppression of T-cell responses through selective induction of TGFβ and T cell apoptosis and activation of tolerogenic circuits on dendritic cells. These glycosylation-dependent functions account for the capability of this lectin to dampen inflammation in autoimmune, chronic and acute inflammatory disorders. Gal-1 is expressed in different portions of the gastrointestinal tract, and has been implicated in different intestinal disorders.

*Yersinia enterocolitica* (Ye) is a Gram-negative, predominantly extracellularly located pathogen that causes foodborne acute or chronic gastrointestinal diseases. During the course of an infection with *Yersinia*, the bacteria colonize the intestinal tract, enter through the M cells of PP, colonize the PP and may eventually disseminate to the MLN, and subsequently, to spleen, liver, and lung. The role of Gal-1 in Ye infection has not been fully explored yet.

**Materials and Methods**

**Objective**

The purpose of the present work was to investigate the role of Gal-1 in the mucosal immune response against oral infection with Ye.

**Results**

**Analysis of PP and MLN cellular populations by flow cytometry**

**Conclusions**

We conclude that Gal-1 may be involved in M cell development and in the control of mucosal immune response after Ye infection. The absence of Gal-1 may favor CD8 T cell response which could act to maintain protective immune response contributing to Ye eradication.