

Name: Paula Guidry

Email: paula.guidry@utsouthwestern.edu

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Title: Non-classical class I MHC in the gastrointestinal tract

The small intestine is simultaneously an essential site of nutrient absorption, host to a diverse commensal microflora, and the first line of defense against a multitude of oral pathogens. As such, it must temper innate and acquired immune responses with tolerance to oral antigens and normal flora. Key players in this immunological balancing act include TCR $\alpha\beta$ and TCR $\gamma\delta$ intestinal intraepithelial lymphocytes (iIEL) and a variety of immune cells in the lamina propria. Many of the gut immune effectors (T and NK cells) are thought to interact with MHC class I or class I-like molecules. We sought to identify a subset of MHC class Ib that may regulate gut mucosal immunity. Transcription of multiple Q, T and M genes was detected in the intestinal tissues of several mouse strains. One of these genes, H2-BI, which was previously thought to be expressed only in the immune-privileged blastocyst and placenta, was selected for further study. We sequenced H2-BI cDNA from the small intestines of several mouse strains and wild mice. To date we have found three major alleles of H2-BI which differ both in primary sequence and splicing variants produced, the first known MHC class I molecule to demonstrate splicing polymorphisms. The common feature of these splicing variants is a Qdm-like leader; Qdm has been reported to interact with the NK cell inhibitory receptor CD94/NKG2A through association with Qa-1. These findings suggest that H2-BI may play an immunoregulatory role in the small intestine.