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Invitation to the "PaP" PhD Seminar

We would like to kindly invite you to the presentation of **Prof. Gianluca Matteoli**, Department of Clinical and Experimental Medicine, Faculty of Medicine, KU Leuven.

Date: **6 December 2018, Thursday**

Time: **5:00 p.m. (s.t.)**

Room: **lecture hall M**

Title: **"Enteric nervous system and myeloid cells crosstalk in homeostasis and inflammation"**

Abstract:

Patients affected by intestinal inflammation suffer from episodes of intestinal dysmotility referred to as ileus. Current view is that ileus depends upon a local inflammatory response mediated by myeloid cells migrating into the gut muscularis externa leading to neuromuscular dysfunction.

However, recent studies have shown that recruitment of leukocytes, and in particular monocytes, is crucial to the timely resolution of inflammation and to prevent excessive tissue damage. To clarify whether monocytes mediate tissue damage and intestinal dysfunction or they are rather involved in the recovery of gastrointestinal motility, intestinal inflammation was induced in C-C motif chemokine receptor 2-deficient (Ccr2-/-) and wild-type (WT) mice via intestinal manipulation (IM). Notably, gastrointestinal motility and transit time after IM were significantly delayed in Ccr2-/- mice compared to WT mice, associated with increased neutrophil-mediated immunopathology and impaired neuromuscular function. Lack of monocyte recruitment to the gut wall resulted in severe functional and morphological alterations at level of the enteric nervous system, suggestive of monocyte neuro-supportive functions during inflammation. In line, molecules secreted by monocyte-derived macrophages promoted neuronal and glial proliferation and differentiation in primary cultures of enteric neural ganglia. Our study reveals a critical role for monocyte-derived macrophages in supporting enteric neuronal functions and restoring functional homeostasis upon intestinal inflammation. From a therapeutic point of view, our data indicate that inappropriate targeting of monocytes may increase inflammation-induced intestinal dysmotility and prolong the clinical outcome of ileus, while future therapies should be aimed at enhancing MΦ repair functions.