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### ► **To cite this version:**

Ambre Riba, Corinne Lencina, Valérie Alquier-Bacquié, Cherryl Harkat, Marion Gillet, et al.. Early maternal separation leads to abnormal immune response against commensal microbita in adult mice. 9. European Mucosal Immunology Group Meeting (EMIG), Oct 2014, Glasgow, United Kingdom. hal-01601550

**HAL Id: hal-01601550**

**<https://hal.science/hal-01601550>**

Submitted on 5 Jun 2020

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## EARLY MATERNAL SEPARATION LEADS TO ABNORMAL IMMUNE RESPONSE AGAINST COMMENSAL MICROBITA IN ADULT MICE

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**1821/ 2500 espaces compris (248/250 words)**

Neonatal period is characterized by immature intestinal epithelium and immune system. Occurrence of adverse events during this period induces long lasting alterations of intestinal homeostasis associated with susceptibility to develop gastrointestinal disorders at adulthood. To characterize the adverse effect of early stress on immune system development, we analyzed the consequences of maternal separation (MS) in mice on intestinal barrier functions and systemic immune response toward microbiota.

In 50-days old male mice, MS increased intestinal permeability to FITC-Dextran-4kDa and visceral sensitivity. MS decreased fecal lysozyme activity, IgA concentration and ILC3 population. This breach in mucosal barrier function was associated with a systemic IgG response against *E. coli*. Furthermore, MS increased anti CD3/CD28-induced IFN $\gamma$  and IL10 secretion in splenocytes. These results are in accordance with an increase of CD4<sup>+</sup>CD44<sup>high</sup>CD62L<sup>low</sup> active T cells and decreased CD4<sup>+</sup>CD25<sup>+</sup>Foxp3<sup>+</sup> regulatory T cells. Furthermore, MS decreased CD40 expression in CD11c<sup>+</sup> and CD11b<sup>+</sup> leading to decrease of IL10 and TGF $\beta$  secretion without any modification of IFN $\gamma$  on *E. coli*-stimulated splenocytes suggesting a defect of antigenic presentation. No modification (quantitative nor qualitative) of fecal *E. coli* population was observed at 50 days in MS mice.

For the first time, we demonstrated that early stressful events impaired intestinal barrier functions leading to an exacerbated humoral response toward microbiota. Inflammatory T cell response was increased in MS mice but dampened by an antigenic presentation defect. Adult mice experiencing early stressful events mimic common symptoms of gastrointestinal disorders and might be an important factor in those pathologies.

Keywords: MS, humoral response toward microbiota, antigen presentation defect, pro-inflammatory T cell response