

## **Supplemental Material**

# **Autophagy: A Novel Mechanism Involved in the Anti-Inflammatory Abilities of Probiotics**

Mazen Zaylaa<sup>a,b</sup> Jeanne Alard<sup>a</sup> Imad Al Kassaa<sup>b</sup> Veronique Peucelle<sup>a</sup>  
Denise Boutillier<sup>a</sup> Jérémie Desramaut<sup>a</sup> Philip Rosenstiel<sup>c</sup> Hang T. T. Nguyen<sup>d</sup>  
Fouad Dabboussi<sup>b</sup> Bruno Pot<sup>a</sup> Corinne Granette<sup>a</sup>

<sup>a</sup>University of Lille, CNRS, Inserm, CHU Lille, Institut Pasteur de Lille, U1019 - UMR 8204 - CIIL - Center for Infection and Immunity of Lille, Lille, France, <sup>b</sup>Laboratoire de Microbiologie Santé et Environnement (LMSE), Doctoral School of Sciences and Technology, Faculty of Public Health, Lebanese University, Tripoli, Lebanon, <sup>c</sup>University of Kiel, University Hospital Schleswig Holstein, Kiel, Germany, <sup>d</sup>M2iSH, Inserm U1071, INRA USC 2018, Université Clermont Auvergne, Clermont-Ferrand, France

## **Supplementary Figure legends**

**Figure S1.** PBMCs (A) or BMDCs (B) pretreated or not for 1h with 3-MA or *atgl16l1*-deficient (KO) BMDCs were stimulated with the bacteria at a bacteria/cells ratio of 10:1 or with Rapamycin (10 µg/ml) for 4 h. The autophagy was followed by immunoblot analysis using antibodies against LC3 to analyze the LC3-II conversion.

