The profile of dendritic cell and T cell response is related to the viral trigger in children with severe asthma exacerbation

Dr. Clémence 10796 Mordacq clémence.mordacq@chru-lille.fr MD 1,2, Dr. Antoine 10797 Deschildre antoine.deschildre@chru-lille.fr MD 1,2, Prof. Isabelle 10798 Tillie-Leblond isabelle@chru-lille.fr MD 1, Dr. Anny 10799 Dewilde anny.dewilde@chru-lille.fr MD 3, Dr. Muriel 10800 Pichavant muriel.pichavant@pasteur-lille.fr 1, Dr. Caroline 10801 Thumerelle caroline.thumerelle@chru-lille.fr 2, Dr. David 10806 Romero drdavid79@yahoo.com MD 2 and Dr. Philippe 10807 Gosset philippe.gosset@pasteur-lille.fr 1. 1 CIIL-LI3- INSERM 1019 - CNRS UMR 8204, Institut Pasteur de Lille, France, 59000 ; 2 Pneumologie et allergologie Pédiatriques, Hôpital Jeanne de Flandre, CHRU Lille, France, 59037 and 3 Laboratoire de Virologie, CHRU Lille, Lille, France, 59037.

Viral infection are associated with asthma exacerbations (AE). Activation of dendritic cells (DC) plays a key role in the response to virus and drives the activation and polarization of T cells. Mobilization of Pattern Recognition Receptor (PRR): Toll Like Receptor (TLR3), RNA helicases (RIG-I, MDA-5) are involved. Our purpose was to analyze expression and function of the PRR in DC and to corroborate this with the presence of virus and the T cell response. Methods: 54 allergic asthmatic children (6-15 y) included during hospitalization for severe AE. Virus identified on nasal secretions by RT-PCR. T cell response determined in blood and induced sputum at the inclusion and in the stable state, 8 weeks later. Mononuclear cells (MNC) stimulated in vitro with poly(IC) and liposomes containing poly(IC) and levels of IL-4, IFN-g, IL-17A measured. Expression of markers of maturation (CD80, CD86) and PRR studied in circulating DC by flow cytometry. Results: Virus were indentified in 60 % (Rhinovirus: 82 %). A Th1 and Th17 (IFNg, IL17A) response was observed in the airways and the blood from the infected patients (V+) during exacerbation whereas a Th2 (IL-4) response prevailed in non-infected patients (V-). The stimulation of MNC induced a Th2 and Th17 response for V+ at inclusion, but Th1 in V-. A defect in RNA helicases expression by blood DC was observed in V+ at inclusion, while the expression of the markers of maturation did not differ among both groups. Conclusion: Viral infection modifies the T cell response during AE and is associated with a defect of RNA-helicase expression in DC. This could contribute to describe new mechanisms in the virus induced AE.