D45
Viral status in asthmatic preschool children during a severe exacerbation: the VIRASTHMA 2 study
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Introduction
During an exacerbation of asthma in preschool children (<5 years), viruses, especially rhinoviruses (hRV) are the main triggers of an inflammatory process, leading to clinical symptoms. Previous works have formulated the hypothesis that these patients have a deficient innate immune response to these pathogens, enabling reinfection. The main purpose of the VIRASTHMA 2 study is to describe the inflammatory profile during and after the resolution of a severe exacerbation. This work focuses on microbiological status.

Methods
Multicentric prospective study in the Haut-de-France region (France). Asthmatic children aged 1 to 5 years, were included during a hospitalization for a severe exacerbation. Clinical history, atopic status, viral status (PCR in a nasal swab sample, hRV typing by amplification of the viral protein (VP) 2/VP4 region), bacteriological status (culture of an induced sputum) were assessed during the exacerbation and at steady state, 8 weeks later. We describe the first 105 patients.

Results
During exacerbation, a virus was identified in 93% of cases, a hRV in 74% (R+ patients), an enterovirus in 13%, an adenovirus in 11%, a respiratory syncitial virus in 7%, a viral co-infection in 27%. Among R+ patients, hRVC was found in 77%, hRVA in 23%, no hRVB was found. We observed a higher median PRAM severity score at admission in R+ patients versus patients infected with another virus (6 vs 4, p=0,004) but no difference in the median length of hospitalization. There was no difference in the prevalence of severe intermittent asthma (26% vs 24%, p=1), a trend toward a higher prevalence of atopy (positive prick tests and/or specific IgE) in R+ patients (59% vs 31%, p=0,053). Among the 67 performed bacteriological cultures, 60% were positive, identifying Haemophilus influenzae (n=25), Moraxella catarrhals (n=20), and Streptococcus pneumonia (n=12). In all, 37 patients (55%) had a viral/bacterial co-infection. At steady state, 52% were R+, hRVA in 65%, hRVC in 23%, hRVB in 12%. Among these patients, only 28% had clinical signs of viral infection. In all, 35% of patients were R+ at exacerbation and at steady state, none of them were infected with the same hRV type at both times.

Conclusion
We confirm a high prevalence of hRV infection, especially hRVC, during exacerbation of asthma in young children, frequently associated with a bacteriological carriage or infection. At steady state, virus carriage was frequently observed. Exploring the host immune innate responses could help better understanding the pathogenic role of these microorganisms.