28th ECCMID EUROPEAN CONGRESS OF CLINICAL MICROBIOLOGY AND INFECTIOUS DISEASES
Madrid, Spain
21 – 24 April 2018

O0525 Effects of the exposure of human corneal epithelial cells and airway epithelial cells of nasal origin to a mixture of four mycotoxins secreted by Aspergillus and Fusarium molds

Septa Pratama[1], Yélian Marc Bossou[1], Sandrine Vitry[2], Isabelle Momas[1], Nathalie Seta[3], Sophie Achard[1], Jean Menotti*[4]

[1]Université Paris-Descartes, EA4064 Environmental Epidemiology Unit, PARIS, France, *[2]Institut Pasteur, Viral Neuroimmunology Unit, PARIS, France*, *[3]Université Paris-Descartes / AP-HP, EA4064 Environmental Epidemiology Unit / Laboratory of Biochemistry of Bichat hospital, PARIS, France*, *[4]Universités Lyon 1 / Paris-Descartes / Paris-Diderot, Laboratory of Parasitology-Mycology of Lyon University Hospitals / EA7426 / EA4064, LYON, France*

Background: Airway exposure to molds and mycotoxins is associated with asthma and chronic airway inflammation. As mold- and mycotoxin-exposure also concerns the eyes, our hypothesis is that mycotoxin exposure on the ocular surface has an impact on the onset of respiratory symptoms. Our aim was thus to study the inflammatory response of Human Corneal Epithelial (HCE) cells exposed to a mixture of mycotoxins secreted by *Aspergillus* and *Fusarium* molds and to assess the role of inflammation markers produced by the HCE cells after mycotoxin exposure on the inflammatory response of human Airway Epithelial Cells of Nasal origin (hAECN).

Materials/methods: The concentrations of four mycotoxins (aflatoxin B1, gliotoxin, deoxynivalenol and fumitremorgin C) which induced a mild increase of inflammatory response and no significant decrease on cell viability were chosen to perform mixtures of the four mycotoxins. Cell viability was measured with a MTT test. Noninvasive label-free real-time cell monitoring was performed by using impedance measurement with xCELLigence plates. The dosage of IL-8 production was performed with ELISA kits. The gene expression of seven proinflammatory cytokines was quantified by real-time reverse transcription PCR.

Results: HCE cell viability decreased from 64 µg/ml aflatoxin B1, 500 ng/ml gliotoxin and 500 ng/ml deoxynivalenol. The production of IL-8 increased from 16 µg/ml aflatoxin B1, 250 ng/ml gliotoxin, 1000 ng/ml deoxynivalenol and 24 µg/ml fumitremorgin C. The mixtures of mycotoxins induced the gene expression of IL-8 and CXCL-1 inflammation markers in HCE cells. After 48h of contact of hAECN with HCE cells exposed to the mixture of 4 mycotoxins, an increase in ICAM-1, IL-23α, IL-8, and TNF-α gene expression was observed. Cell impedance measurements showed an inhibition of hAECN cell proliferation after co-exposure with mycotoxin-exposed HCE cells.

Conclusions: This study showed a potentiation of the inflammatory response by the exposure of HCE to a mixture of mycotoxins, which is of public health interest as occupational exposure to mixtures of mycotoxins is frequent in agricultural settings for example. Also, we showed a proinflammatory and anti-proliferative effect of mycotoxin-exposed HCE cells on hAECN, underlying the need to protect the eye in addition to the nose for mycotoxin-exposed workers.