SIM
SOCIETÀ ITALIANA
DI MICROBIOLOGIA

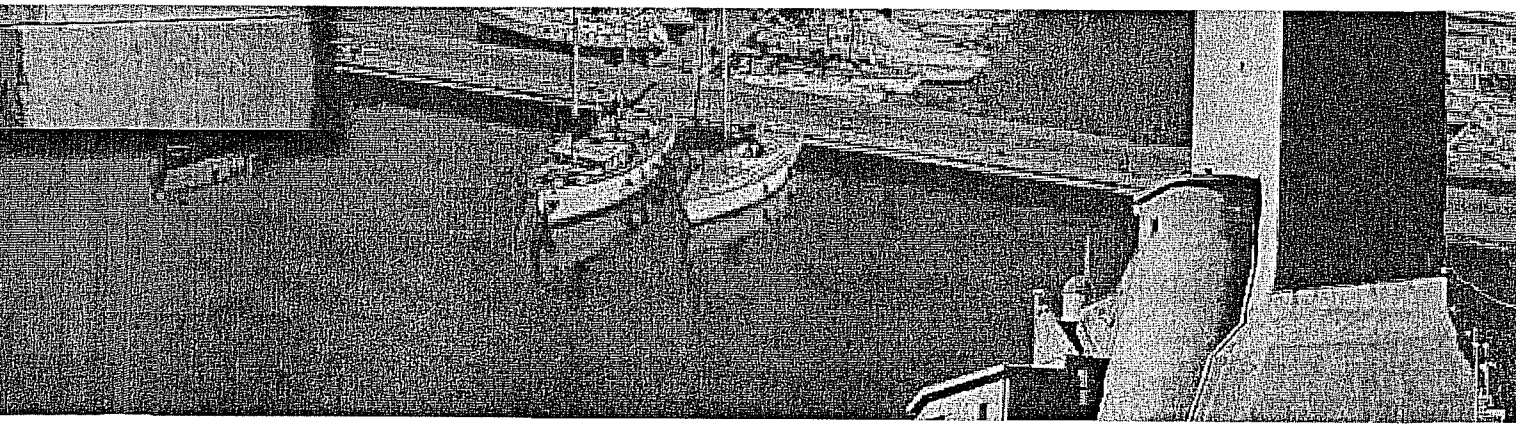
45° CONGRESSO NAZIONALE DELLA SOCIETÀ ITALIANA DI MICROBIOLOGIA

SIM 2017

Genova, Palazzo Ducale

27 - 30 settembre 2017

Abstract Book



P68

INFLAMMATORY RESPONSE DURING HUMAN VAGINAL INFECTION WITH *CANDIDA ALBICANS*

Elena Roselletti[1], Stefano Perito[1], Elena Gabrielli[1], Eva Pericolini[2,3], Samuele Sabbatini[1], Anna Vecchiarelli[1]

[1]Department of Experimental Medicine, University of Perugia, Perugia - Italy
[2]Department of Diagnostic, Clinic and Public Health Medicine, University of Modena and Reggio Emilia, Modena-Italy [3]Department of Experimental Medicine, University of Perugia, Sant'Andrea delle Fratte, Perugia - Italy

Introduction: The microbiological, pathological and clinical factors determining vaginal candidiasis and recurrent vaginal candidiasis have long been studied, particularly using rodent models. The validity of which for understanding the pathogenesis of disease in women has been questioned. The most prevalent agent is critically determined by activation of microbial and host factors leading to persistent vaginal inflammation coupled to the inability of the inflammatory cells to resolve the fungal infection. Here we studied the activation of inflammasome complex neutrophil-recruiting and activating cytokines in the vaginal secretion of women with clinically established vaginal candidiasis.

Materials and Methods: In human vaginal samples positive for *C. albicans* with vaginal candidiasis (n = 20) and carriage (n = 15), infiltration of neutrophils ,inflammatory mediators such as IL-8 and IL-1β, activation of inflammasome complex and expression of aspartyl proteases (SAPs) were examined.

Results: In vaginal swabs of patients with vaginal candidiasis we found: i) consistent recruitment of neutrophils; ii) appreciable level of IL-8 and IL-1β; iii) activation of inflammasome complex; iv) consistent expression of *SAP2, SAP5* and *SAP6.*

Conclusions: These results show that immunopathogenesis of vaginal candidiasis is mediated by local recruitment of neutrophils, inflammatory cytokines secretion and inflammasome activation that mirror the upregulation of *SAP2, SAP5* and *SAP6* gene expression.

P69

BETA-DEFENSIN-2 AND -3 REDUCE INTESTINAL DAMAGE CAUSED BY *SALMONELLA TYPHIMURIUM* INFECTION

Alessandra Fusco[1], Anna De Filippis[1], Brunella Perfetto[1], Pina Caputo[1], Chiara Schiraldi[1], Giovanna Donnarumma[1]

[1]Department of Experimental Medicine, section of Microbiology and clinical Microbiology, University of Campania Luigi Vanvitelli, Naples - Italy

Introduction: The human intestine hosts a large and diverse microbial community estimated to contain approximatively 400-1000 different species of bacteria, virus and fungi. These microbes are collectively referred to as the microbiota. Among the main functions of the intestinal microbiota are the synthesis of essential amino acids and vitamins (K, B2, B1, folic acid, biotin, and pantothenic acid) and extraction of energy from components in the diet as some are not digestible polysaccharides of plant origin. Moreover, it is involved in the absorption of calcium, magnesium and iron, and contributes to maintaining the integrity of the intestinal wall, modulating responses to pathogenic noxae, and representing a key factor in the maturation of the immune system. In fact, it plays an active role in the intestinal immune response through the secretion of inflammatory cytokines, chemokines, and antimicrobial peptides such as b-defensins. Between these, human beta-defensin-2 and -3 (hBD-2 and hBD-3) are expressed at mucosal sites and exhibit broad antimicrobial activity against Gram-positive and Gram-negative bacteria, mycobacteria, fungi, and viruses. In addition to direct antimicrobial properties, hBD-2 and hBD-3 recruit innate and adaptive effector cells to sites of inflammation, induce cytokines and mast cell degranulation, and aid in wound healing. The aim of this study it was to create a line of intestinal epithelial cells expressing high concentrations of the antimicrobial peptides hBD-2 and hBD-3, and to assess their role in the host inflammatory response resulting from bacterial infections.

Materials and Methods: *Cloning and transfection*: genes coding for antimicrobial peptides HBD-2 and HBD-3 were cloned into the pEF/V5-HIS TOPO vector then transfected into Caco-2 cells previously subcultured for 21 days to obtain their full differentiation. *Infection and Real-Time PCR:* the transfected cells were subsequently infected with *S. typhimurium* and the modulation of expression