



47° CONGRESSO NAZIONALE

SOCIETÀ ITALIANA DI MICROBIOLOGIA

18-21 SETTEMBRE 2019
ANGELICUM CONGRESS CENTRE

Pontificia Università San Tommaso d'Aquino
Largo Angelicum 1 – 00184 Roma



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P 129 - ID 012 - Atopobium vaginae and Porphyromonas somerae induce proinflammatory cytokines expression in endometrial cells: a possible implication for endometrial cancer?

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Introduction: The female lower vaginal tract has long been known to have an active microbiota, but a uterine microbiota was recognized only recently, with unique characteristics differing from that of the vagina. Although the exact role of uterine microbiota is unclear, recent studies suggest that it could affect important conditions including endometriosis and cancer. In particular, *Atopobium vaginae* and *Porphyromonas somerae* species resulted particularly significant in endometrial cancer, but there is currently uncertainty about the mechanisms by which such bacteria might influence endometrial microenvironment. Our study was thus aimed to analyze *in vitro* the effect of *A. vaginae* and *P. somerae* on the expression of pro-inflammatory cytokines by endometrial cells, as they are implied in the establishment of an inflammatory environment potentially favoring the onset/progression of endometrial cancer.

Materials and Methods: The human HEC-1A endometrial cells were cultured in the presence or absence of *A. vaginae*, *P. somerae* or *Lactobacillus vaginalis* (as a control). Cells and cell supernatant were collected at 24, 48 and 72 hours post infection (h.p.i.), and analyzed for proinflammatory cytokine expression and release by ELISA and transcription analysis.

Results: *A. vaginae* and *P. somerae* induced an evident release of proinflammatory cytokines by endometrial cells, with different patterns for *A. vaginae/P. somerae* (IL1alpha, IL1beta, IL17alpha, and TNFalpha) compared to *L. vaginalis* (IL-8). Cytokine mRNA expression analysis, performed by microarray real-time PCR, showed that all the cytokines detected by ELISA were activated at the transcriptional level. In addition, other chemokines resulted significantly altered by co-culture with *Atopobium* and *Porphyromonas*, including CCL8, CXCL2, IL22 and IL9, all of them previously reported to be associated with cancer progression. In particular, CCL8 has been associated with tumor progression and dissemination, CXCL2 is induced by TNFalpha and induces chemoresistance, IL22 promotes progression in breast cancer, and IL9 prevents apoptosis, promoting proliferation and metastasis.

Discussion and Conclusions: Our *in vitro* observations show for the first time that certain bacteria have the ability to induce expression of proinflammatory cytokines and chemokines by endometrial cells, and suggest that their presence in the uterine environment might be associated with the establishment of conditions promoting inflammation, providing a starting point for future researches focusing on the impact of uterine microbiota on uterine physiology and pathologies, and hopefully potentially useful tools for diagnosis and effective clinical interventions.