Abstract form 8th ESIDOG Conference London October 24th – 27th 2013

Title: **Can a specific sub-group of biofilm- forming Gardnerella vaginalis strains be the real causative agent of bacterial vaginosis?**

Authors: **Castro J[1], Machado A[1,2], Alves P[1], Sousa C[1], Cereija TB[1], França A[1], Jefferson KK [2]**and **Cerca N[1*]**

Affiliation: [1]Institute of Biotechnology and Bioengineering - CEB-IBB - University of Minho, Braga, Portugal
[2]Department of Microbiology and Immunology, Virginia Commonwealth University, Richmond, VA 23298-0678, USA
* Correponding author: nunocerca@ceb.uminho.pt

Preferred type of presentation X Oral communication (12min+3) ◘ Electronic poster (5min +2)

Data presented ◘ Preliminary results X Data in / not yet published X Published

Session X Sexual Transmitted Infections ◘ Vulvovaginitis X Infections in pregnancy ◘ HPV ◘ Other

Introduction:

In the past half century, bacterial vaginosis (BV) has been a controversial topic in medical microbiology, and despite the wealth of information on this topic, the etiological agent has not yet been definitively identified. A couple of years ago the first descriptions of multispecies biofilm communities were described in BV. Interestingly, *G. vaginalis* was present in most cases and accounted for the majority of the biofilm biomass.

Aim:

We tested the hypothesis that strains of *G. vaginalis* that were able to form biofilms could be the causative agent of BV.

Methods:

We isolated more than 30 bacterial species from BV patients and also several strains of *G. vaginalis* from healthy women, and tested biofilm forming ability, initial adhesion to human vaginal cells, cytotoxicity activity, antimicrobial resistance and gene expression of know virulent genes.

Results:

Our results revealed that *G. vaginalis* outcompeted all the other bacterial species in the initial adhesion to the epithelial cells. Furthermore, when comparing BV-associated *G. vaginalis* strains to strains isolated from healthy women, we found that all 7strains from BV were more virulent than the 7 strains colonizing healthy women, as measured by the higher cytotoxicity and the higher initial adhesion to epithelial cells. No significant differences were found in antimicrobial resistance profiles. Interestingly, no significant differences in expression of known virulence genes were detected, suggesting that the higher virulence of the BV-associated *G. vaginalis* was due to a yet unknown virulence determinant. We then tested virulent *G. vaginalis* against other known BV-associated anaerobe pathogens, in mixed biofilm formation quantification. Interestingly, while the other tested anaerobes did not reveal a higher initial adhesion, they did enhance biofilm formation by *G. vaginalis*.

Conclusion:

Our data suggests that virulent variants of *G. vaginalis* have the potential to be the etiological agent of BV, while acknowledging that other anaerobes do enhance *G. vaginalis* virulence.