Impact of estradiol on biofilm formation of Pseudomonas aeruginosa clinical isolates from cystic fibrosis patients

Faria Afzal¹, Tony Le Gall², Tristan Montier², and Mareike Müller¹

¹Physical Chemistry I & Research Center of Micro and Nanochemistry and Engineering (Cμ), Department of Chemistry and Biology, University of Siegen, Siegen, Germany (m.mueller@chemie-bio.uni-siegen.de)
²“Gene Transfer and Gene Therapy” Team, INSERM UMR 1078; IBSAM; Laboratoire de Génétique Moléculaire et Histocompatibilité, CHRU Brest; UFR Médecine et Sciences de la Santé, Brest, France

Women with Cystic Fibrosis (CF) have a significantly lower life expectancy compared to men, which is indicated by an earlier impairment of lung function due to chronic colonization of pathogenic bacteria like Pseudomonas aeruginosa (PA). Reasons for this “CF gender gap” until now have not yet been fully clarified and are assumed to be multifactorial.

This study aims to shed light on the contribution of sex hormones to the CF-Gender gap considering microbial endocrinology. Therefore, the study investigates whether the sex hormone estradiol, whose blood serum concentrations are significantly fluctuating within the female cycle and during pregnancy, has a regulatory influence on the development of PA biofilms in the context of CF.

For that purpose, biofilms of PA isolates from CF-patients are treated in vitro with various estradiol concentrations and are examined in a comparative study quantitatively regarding the total biomass, e.g. via crystal violet staining, and qualitatively, e.g. via scanning electron microscopy, to characterize the ultrastructure of the biofilm.

We observed that estradiol induces biofilm-forming capacity of CF-PA-isolates with respect to the total biomass and modulates the biofilm structure especially concerning the distribution and clustering of bacteria.

The observed in vitro correlation between estradiol concentration and extent of biofilm growth provides a possible microbiological explanation for gender differences in CF disease progression.

These insights and further research on possible underlying mechanisms might be relevant in the long-term for new approaches in personalized treatment for female CF patients.

Acknowledgement

This work is supported by a financial grant from Mukoviszidose Institut gGmbH, Bonn, the research and development arm of the German Cystic Fibrosis Association Mukoviszidose e.V., the Christiane Herzog foundation. We further thank the Equal Opportunities Office of the University Siegen as well as the DAAD PPP Frankreich (Project-ID 55976814) for financial support.