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Basic science: biofilm pathophysiology

ANTI-BIOFILM ACTIVITY SECRETED BY STAPHYLOCOCCUS AUREUS AGAINST STAPHYLOCOCCUS EPIDERMIDIS

M. Tilotta[1], R. Papa[1], A. Cellini[1], L. Selan[1], M. Artini[1]

[1]Public Health And Infectious Diseases, Sapienza University, Rome, Italy

Objectives

In medical settings, biofilms are the cause of persistent infections-implicated in 80% or more of all microbial cases releasing harmful toxins and even obstructing indwelling catheters or causing orthopaedic implant infections. Staphylococci are recognized as the most frequent causes of biofilm-associated infections, thanks to their ability to adhere on both eukaryotic cells and abiotic surfaces via cell wall. Staphylococcal biofilms display extraordinary resistance to antimicrobial killing and limit the efficacy of antibiotic therapy; thus surgical intervention is required to remove medical devices. *Staphylococcus aureus* and *Staphylococcus epidermidis* are prevalent species on skin and mucosae of animals and humans. The virulence of staphylococci is due to the combined effect of extracellular factors, toxins (only *S. aureus*), biofilm formation and resistance to phagocytosis.

The interest in the development of alternative anti-infective approaches for the prevention and treatment of infections increased in recent years. Our rationale is to look for new antimicrobials inhibiting virulence, in particular bacterial adhesion and biofilm formation, rather than bacterial growth. This latter should exert a weaker selective pressure for the development of drug resistance.

Methods

In this work we examined the anti-bio?lm activity of cell-free supernatants deriving from planktonic and sessile growths of *S. aureus* and *S. epidermidis*. Their effect on staphylococcal biofilm formation was evaluated both in static and dynamic condition. The dynamic condition was assessed in BioFlux System, a device based on microfluidics that precisely controls the flow of growth medium between two interconnected wells of a microtiter plate, in order to acquire sequential bright-field images of any growing biofilm.

Results

Data obtained have shown the anti-biofilm activity of the supernatants of *S. aureus* able to inhibit the biofilm formation, and to disrupt mature biofilms of *S. epidermidis* without affecting the bacterial viability. The anti-biofilm activity was present in supernatants obtaining in planktonic and sessile growth conditions. Subsequently some physico-chemical treatments were performed in order to define the nature of anti-biofilm compound(s). Treatment with protease eliminated anti-biofilm activity in supernatant deriving from sessile cultures while it did not affect the anti-biofilm activity in supernatant deriving from planktonic cultures. Therefore anti-biofilm activity can be due to molecules or complexes differentially expressed in the two growth conditions (planktonic and sessile). The identification of the active molecule(s) is a prerequisite for the understanding of the mode of action.

Conclusion

These findings suggest the possibility to identify anti-biofilm compounds obtained from *S. aureus* cell-free culture supernatants, grown in both planktonic and sessile condition.