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**MICROBIOLOGY & MOLECULAR GENETICS**

**Departmental Journal Club**

**MICR 6120**

**Monday**

**October 17th, 2016**

11:30am-12:20pm

RM 122 Classroom Bldg.

Presented by

Khadija Abdulhafid
PHD Student

Title: The Quorum Sensing Inhibitor Hamamelitannin Increases Antibiotic Susceptibility of Staphylococcus aureus Biofilms by Affecting Peptidoglycan Biosynthesis and eDNA Release

Authors: Gilles Brackman, Koen Breyne, Riet De Rycke, Arno Vermote, Filip Van Nieuwerburgh,

Evelyne Meyer, Serge Van Calenbergh & Tom Coenye

Treatment of Staphylococcus aureus infections has become increasingly challenging due to the rapid emergence and dissemination of methicillin-resistant strains. In addition, S. aureus reside within biofilms at the site of infection. Few novel antibacterial agents have been developed in recent years and their bacteriostatic or bactericidal activity results in selective pressure, inevitably inducing antimicrobial resistance. Consequently, innovative antimicrobials with other modes of action are urgently needed. One alternative approach is targeting the bacterial quorum sensing (QS) system. Hamamelitannin (2′,5-di-O-galloyl-d-hamamelose; HAM) was previously suggested to block QS through the TraP QS system and was shown to increase S. aureus biofilm susceptibility towards vancomycin (VAN) although mechanistic insights are still lacking. In the present study we provide evidence that HAM specifically affects S. aureus biofilm susceptibility through the TraP receptor by affecting cell wall synthesis and extracellular DNA release of S. aureus. We further provide evidence that HAM can increase the susceptibility of S. aureus biofilms towards different classes of antibiotics in vitro. Finally, we show that HAM increases the susceptibility of S. aureus to antibiotic treatment in in vivo Caenorhabditis elegans and mouse mammary gland infection models.