CALIFORNIA STATE UNIVERSITY, LONG BEACH

COLLEGE OF NATURAL SCIENCES AND MATHEMATICS

FACULTY RESEARCH SYMPOSIUM 2016

Eighth Annual CNSM Faculty Research Symposium

Friday, March 11, 2016

Call for Abstracts

Abstracts due: Friday, February 12, 2016

Email to: Margaret.Karteron@csulb.edu

Abstract Submission Guidelines: *see next page.*

All CNSM Faculty are invited.

Mark your calendars for:

Friday, March 11, 2016

12:30 – 5:30 p.m.

CSULB University Student Union Ballroom

GUIDELINES FOR ABSTRACT SUBMISSION
CNSM Faculty Research Symposium 2016

Note: email an electronic copy of your abstract in MS Word format to Margaret.Karteron@csulb.edu by Friday, February 12, 2016. Indicate your preference for a poster or a platform presentation in your cover email.

Abstracts will be published in the symposium program as well as published on the College website, so please meet the following formatting guidelines:

HEADER (see example on the next page)

- **Title** – The entire title is written in bold and the first letter of each word is capitalized. Only scientific names are written in *italics* with just the first letter of the genus capitalized.
- **Presenters** – The first person listed is the faculty member presenting. The presenter's name is underlined.
- **Authors** –If the authors are from more than one department/institution, then a superscript number follows the surname of each author to identify the institution each of the authors is associated with. Please include an asterisk (*) behind the names of student authors. The asterisk should follow the institution identification.
- **Department and institution affiliations** – List the complete name of department; full name of institution; and city, state, and zip code for each author. Please note that the corresponding superscript precedes the department name.

ABSTRACT BODY (see example on the next page)

- The abstract should summarize the purpose of the study, methods used, results and conclusion.
- Limited to 2,500 characters, not including spaces.
- DO NOT include the title and/or author(s) in the abstract block.

GRANT SUPPORT (see example on the next page)

- Acknowledge the granting agency and grant number which provided support for this research. There may be multiple sources of funding.

SAMPLE ABSTRACT WITH CORRECT FORMATTING

Agr Typing and Bacterial Interference in *Staphylococcus aureus*

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Regulation of virulence factor expression in the pathogen, *Staphylococcus aureus*, is coordinated by the *agr* (accessory gene regulation) operon. The *agr* locus codes for the components necessary for quorum sensing. The Agr system triggers the expression of pathogenicity factors when the cell density is high in response to the accumulation of a self-secreted autoinducing peptide (AIP). Due to interspecies variation at the *agr* locus, each strain secretes an AIP that self-activates but completely inhibits Agr activation in heterologous strains. Most non-aureus species produce AIPs that generally inhibit Agr activation in *S. aureus*, leading to a novel type of bacterial interference. To date, four different Agr groups have been identified in *S. aureus*, with intriguing relationships between Agr type and disease pathogenesis. To further our studies along these lines, we have developed a simple assay to determine the Agr type of new *S. aureus* isolates and to test for AIP-specific, cross-inhibition between staphylococcal species. This functional assay depends on the ability of AIP producing strains to activate or inhibit an Agr-specific luciferase or GFP reporter. A collection of clinical *S. aureus* isolates was examined to verify the assay and to explore any functional relationships between Agr type and pathotype. Most of the *S. aureus* strains secreted a substance that activated one and only one of the four *S. aureus*-group-specific tester strains, suggesting that a single functional AIP is secreted by most clinical isolates. Moreover, multiple isolates from the same patient were almost always the same Agr type. Agr type, hemolytic activity, and clinical manifestations were compared. The simplicity of these assays will facilitate future studies to understand the role of Agr biotypes in pathogenesis and explore the phenomenon of *agr*-based bacterial interference between *Staphylococci.*

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