Clinical distribution of *Pseudomonas aeruginosa* and evaluation of its sensitivity against common antimicrobial agents

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Hundreds samples viz. urine, blood, wound, pus and sputum collected from different patients were found to harbour *Pseudomonas aeruginosa* (27%) with a maximum isolation from wound samples (33.33%) and minimum from blood samples (11.11%). The degree of resistance of *Pseudomonas aeruginosa* isolates to different antibiotics like Ceftazidime (30µg), Amikacin (30µg), Imipenem (10µg), Ciprofloxacin (30µg), Tetracycline (30µg), Gentamicin (10µg), Norfloxacin (10µg), Penicillin (30µg), Chloramphenicol (30µg), and Ofloxacin (5µg) varied from 56% to 100%. Antiseptics (Betadine and Dettol) were found to be effective against the MDR strains of *Pseudomonas aeruginosa* at the dilutions of 10⁻¹ and 10⁻². Duration of the disease and hospitalization duration, evaluated as risk factors for *Pseudomonas aeruginosa* colonization were found to be statistically significant while age and gender were found to be statistically non-significant. The incidence of multidrug resistance of *Pseudomonas aeruginosa* is increasing fast due to the frequent use of antibiotics and antiseptics, which are used extensively in hospitals and healthcare centers, therefore it is a need to develop alternative antimicrobial agents for the treatment of infectious diseases.

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To investigate the biofilm forming genes involved in MRSA and MSSA strains

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The icaADBC was first identified in *S. epidermidis*, and is also present in *S. aureus* and other staphylococcal species. PIA is produced by the gene products encoded by the icaADBC operon. Asymptomatically colonized patients and health care workers are the major sources of MRSA in the hospital environment. MRSA-infected patients in burns units are particularly problematic because the big surface area of denuded skin can produce a large inoculum of organisms that can be easily transmitted to other patients via the hands of health care workers. Extensive skin lesions also result in heavy shedders of MRSA. Clinical isolates of *Staphylococcus aureus* can express the icaADBC-encoded polysaccharide intercellular adhesin/poly-N-acetylglucosamine (PIA/PNAG). The icaADBC dependent and independent pathways will be stimulated using different chemicals and level of biofilm formation as well as PIA/PNAG level will be assayed. Besides, proteomics and transcriptomics analysis will be performed to get insights in the interaction of various factors of the pathways involved in the biofilm formation in wild type as well as mutant strains. The biofilm development in MRSA is ica independent and involves a protein adhesin(s) regulated by SarA and agr, whereas SarA-regulated PIA/PNAG plays a more important role in MSSA biofilm development in ica dependent pathway. This will lead to the establishment of a comprehensive interactome of biofilm formation.

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