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Journal Name:	Microbiology Research Journal International
Manuscript Number:	Ms_MRJI_41483
Title of the Manuscript:	PSEUDOMONAS AERUGINOSA INFECTION IN HUMANS AND ANTIBIOTIC RESISTANCE
Type of the Article	Review Article

General guideline for Peer Review process:

This journal's peer review policy states that **NO** manuscript should be rejected only on the basis of '**lack of Novelty**', provided the manuscript is scientifically robust and technically sound. To know the complete guideline for Peer Review process, reviewers are requested to visit this link:

(<http://www.sciencedomain.org/page.php?id=sdi-general-editorial-policy#Peer-Review-Guideline>)



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PART 1: Review Comments

	Reviewer's comment	Author's comment (if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)
Compulsory REVISION comments	In the introduction section the author states that the pathogen is a consequence of hospital associated infection. What are the means or sources though which the humans encounter the pathogen? Is it through any sought of medical devices or instruments, any type of apparatus used for treating the patient? What is the prime means through with humans encounter the pathogen? The author in the same section states that the infection is common in ICU and burn units? These are the areas where critical cases are admitted. I would like to know if a primary infection by some other pathogen enhances the scope of <i>Pseudomonas</i> invasion or will <i>Pseudomonas</i> act as primary perpetrator which opens up the cope for secondary infections? Can the author clarify on this point? The author in the same section conveys that the mechanism of resistance to antibiotics in the organism is natural? Can this be a consequence of the genetics of the organisms? Do extra chromosomal elements called as plasmids confer the organism with this trait? Is it a consequence of horizontal gene transfer? Does employment of broad spectrum antibiotics makes the organism resistant to a wide range of anti microbial agents. Can the author add some notes on it? In the pathogenesis section the author claims that the pathogen does not have invasive characters but I would like to know the prominence of various toxins by the organism do they have any role in enhancing the scope of infection. Do they make the organism more virulent? When the organism does not have invasive characters then what makes it more resilient to antimicrobial agents? Is it the genetics of the organisms or does the primary infection by some other organism opens the door for <i>Pseudomonas</i>. Is phospholipase C responsible for the heat stable nature of the organism? Dipicolini acid is usually present in endospore. Are they present in <i>Pseudomonas</i>? Does calcium ion in the cell wall also favor the heat stable nature of the organism? The author states that bacterial proteases interfere with the immunoglobulin efficacy? What are the factors that trigger or activate these proteases? Does the ions present in the bacteria assist in their activation? When a foreign body encounters the immune system acts against it? Will the efficacy of lymphocytes be influenced by the bacteria? Lymphocytes are known for activating lymphokines which in turn trigger MAF (Macrophage activating factors). Will these factors be detrimental or will the organism evade its action because of a variety of components produced by it? Does the author feel that empirical treatment has resulted in the resistance in the pathogen? The author has cited few antibiotics and their efficacy on the organisms. What is the optimum concentration for the elimination of the organisms? This in turn depends on the initial load and the type of antimicrobial agent.	



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Minor REVISION comments		
Optional/General comments		

Reviewer Details:

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