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REVIEW OF ACINETOBACTER BAUMANNII: TAXONOMY, EPIDEMIOLOGY, PATHOGENICITY, AND ANTIMICROBIAL RESISTANCE

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Abstract

The genus *Acinetobacter* are a wide spread in nature, and there are at least 25 different types of them. In the medical field, *Acinetobacter baumannii* is one of the most important species of this genus. It is an opportunistic pathogen and has many virulence factors that make the bacteria capable of causing many hospital-acquired diseases which leads to nosocomial outbreak attendant with rise in death rates. This bacteria has the ability to be resistant to many antibiotics, and the emergence of high levels of multi-drug resistance *A. baumannii* has made it a priority health issue and is considered a serious threat to health care facilities, public health and the elderly. Which requires a tremendous effort to stop this escalation.

Keywords: *Acinetobacter Baumannii*, Multi-Drug Resistance, Hospital-Acquired Infections.

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1.Introduction

The genus *Acinetobacter* was first isolated from soil in 1911 by scientist Beijerinck who called it *Micrococcus calcoaceticus* [1,2] . During the 1940 s of the last century it was referred to known as *Acinetobacter* [3] . The genus *Acinetobacter* includes species which are gram-negative coccobacilli , nonmotile ,strictly aerobic and non-oxidase production , while catalase is produced [4] . On usual laboratory media, it can grow easily and are found in humid environments, including ponds, waste water, moist soil, water treatment plant , Rish farms and sea water [5] . *Acinetobacter* are wide spread in nature, and there are at least 25 different types of them [6] . During the past twodecade, the clinical importance of *A.baumannii* isolates increased due to the injuries it caused among the wounded American soldiers during the war in Iraq and Afghanistan and its high ability to acquire resistance to all antibiotics currently used, as well as their ability to stay for a long time in the hospital environment , which lead to their rapid spread and infection out breaks [7,8] . Also, *A.baumannii* is responsible for many hospital acquired infections, including Pneumonia , associated with the artificial respiration system in patients confined to hospital intensive car units (ICUs) . It can infect the skin, wounds, the respiratory System and digestive system in human [9,10] . *A. baumannii* is characterized by having many virulence factors that contribute to its pathogenicity [11] .

2. *Acinetobacter baumannii*

Medically, it is the most important species of the genus *Acinetobacter* . The species was first isolated from soil and water in (1968) by scientist Baumann , as well as from food samples such as meat and vegetables . It was also isolated from various human pathological samples such as blood , sputum , pleural fluid and urine [12] . *A. baumannii* appears as gram – negative coccobacilli that are non – motile , aerobic , opportunistic pathogens that mainly attack hospitalized patients as they have the ability to resist and survive for a long time throughout hospitals , which enhances their ability to spread [13] . Biochemical characteristics of *Acinetobacter baumannii* shown in table (1) . This type can survive for a long time in dry conditions , for example it lasts 7days on dry washed clothes , 6days on dry filter paper , 3days on Formica, 7days on glass and more than 25 days on cotton . It has the ability to grow at a temperature of 44°C and this is what distinguishes it from other species [14].

Table 1 : Biochemical characteristics of *Acinetobacter baumannii*

Tests	Results
Morphology	Coccobacilli
Motility	Non motile
Hemolysis	-ve
Growth at 37°C , 44°C	+ve
Production of Oxidase	-ve
Production of Catalase	+ve
Utilization of citrate	+ve
Hydrolysis of gelatin	-ve
Oxidative-Fermentative(Glucose, Dextrose)	+ve
Arginine	+ve
Malonate	+ve
Production of urease	-ve
Reduction of nitrate	-ve
Indole	-ve
Simmons citrate	+ve
Fermentation of lactose	-ve

(-ve : Negative , +ve Positive)

3.Current Taxonomy State

According to Bergey's Manual of Systematic Bacteriology categorized the *Acinetobacter* genus with the Neisseriaceae family [15], and one species *Acinetobacter calcoaceticus*. The names of the species have persisted great taxonomic modifications through the years because of the superior knowledge of molecular strategies of the genetic makeup of this set of microorganisms [16]. Recent classifications which appear to have enjoyed wide acceptance amongst bacterial taxonomists have accepted this microorganisms as gamma proteobacteria classified within the order Pseudomonadales and family Moraxellaceae. Therefore, the taxonomical category is as follows; Domain , Phylum, Class , Order, Family, Genus (*Bacteria*, *Proteobacteria*, *Gammaproteobacteria*, *Pseudomonadales*, *Moraxellaceae*, *Acinetobacter*) respectively. Species *Acinetobacter baumannii*, *Acinetobacter calcoaceticus*, *Acinetobacter haemolyticus* and *Acinetobacter lawsonii* are of medical importance [17,18].

4.Epidemiology of *Acinetobacter baumannii*

The epidemiology of *Acinetobacter baumannii* is wide spread and includes infection related to wars, tropical environments, and hospital – acquired infections [19]. It can inhabit soil and water, and other probable reservoirs include lice and food animal [20]. In human, high colonization rates of the skin, throat, wounds, respiratory tract and gastrointestinal tracts, of various degree of importance, were documented in numerous outbreaks [21,22]. In particular, outbreaks among patients in the intensive care unit (ICU). In addition many reports have been indicated cause of outbreaks and nosocomial infections involve ventilator - associated pneumoniae, meningitis, septicemia, bacteremia, urinary tract infections and endocarditis [23,24]. Most epidemiological studies have been described the prevalence of infection with multi-drug resistance *A.baumannii* in various regions of the world, including North America, China, Brazil, Japan and Europe [25]. Several cases have been reported from hospitals in the middle East such as United Arab Emirates, Saudi Arabia, Bahrain and Lebanon [26,27]. Community acquired pneumonia have been reported in Asia and Australia especially during the wet season [28,29]. It is not known what causes high rates of infection with *Acinetobacter* in a particular geographical area, however this may be due to the variation in temperature and humidity that affect the colonized bacteria [30].

5.Pathogenicity

A. baumannii is an opportunistic bacterium that has attracted the attention of specialists due to its high ability to develop resistance against many antibiotics (Multiple Drug Resistance (MDR)), and it has the potential to acquire genetic material from different races as well as its biofilm formation and its great ability to adhere to surface [31]. The cause of pathogenicity of *A. baumannii* is due to its possession of many virulence factors, including (curli fiber, cytotoxic, necrotizing factor, siderophores and aerobactin), in addition to its production of colicin (v), gelatinase, capsule and possession of polysaccharides and the formation of pellicle assay. Also it produces some types of enzymes, including lipolytic enzyme (lipase) and proteolytic enzyme (protease) [32]. Diseases caused by *A. baumannii* are wide spread and in different areas of the human body with varying severity among the infected. These infections include: pneumonia, endocarditis, meningitis, wound and burn injuries, peritonitis, osteomyelitis, arthritis and bacteremia [33,34]. It can also colonize the skin, the nervous system and the eyes, and cause soft tissue infections [35,36], and pose a major risk to hospitalized patients, especially those in the Intensive Care Unit (ICU). Those who are immunocompromised and spend long time in the hospital, and these are exposed to infection with bacteria through hospital equipment or through the care workers by direct contact with the infected patient, and this bacteria is contaminated in different parts of the hospital environment, as it was found on curtains, throat examination blades, door handles, wipers, keys, patient lift equipment, as well as medical equipment. The need to pay attention to disinfecting common equipment, especially catheter and respiratory equipment [37,38].

6.Resistance of *A. baumannii* to antibiotics

A. baumannii have spread throughout the world due to their ability to survive in various environmental conditions and to acquire new genetic factors as a result of: first – mobile genetic elements such as transferable plasmid and integrons. Second – the inherited modification of the autonomous genes by mutations that change the target of the antagonists, which in turn change the self – resistance [39,40]. The presence of such motile genetic elements is of concern, as the acquisition of genes can trigger drastic changes in resistance. Additionally, the ability of *A.*

A. baumannii to confer on plasmids carrying anti – resistance traits , which are likely to spread among other types of bacteria [41,42] . The bacteria exhibit self – resistance to various antigens due to the decrease of the permeability in the outer membrane and flow pumps . The bacteria have become resistance to nearly all commonly used antibiotics , including ,B- lactams , Quinolones , polymyxins , Aminoglycosides , Tetracyclines , Chloramphenicol and Glycylcyclines[43,44] . Resistance of *A. baumannii* to beta lactams includes several mechanisms :(A):Enzymatic mechanisms or the production of antigen degradation enzymes . The main mechanisms in resistance to beta– lactam lies in their composition of broad spectrum betalactamases , which are encoded by genes carried on a plasmid or chromosome . The AMPC – B – lactamases is one of the chromosomal enzymes that degrade broad – spectrum cephalosporins , except for cefepime[45,46] . AS well as the enzyme Oxacillinases (OXA),which are characterized by their effectiveness by analyzing anti-carbapenems . As for the enzymes containing minerals , they degrade all B- lactams except for Aztreonam , which are often encoded by mobile genetic elements that can pass easily between bacteria [47] . (B) :Non – enzymatic mechanisms , including changing the permeability of the membrane , either by losing or decreasing outer membrane proteins (OMPs) , or by increasing the flow pumps , which leads to preventing the entry of the antagonist or enhancing its expulsion to the outside [48] . The sequences of penicillin binding proteins (PBPs) varied, as changes occur in the expression levels of normal PBPs . Studies have indicated that there are several differences in the expression pattern of the binding proteins between *A. baumannii* susceptible bacteria and anti – carbapenem resistance , and that decrease in antagonist affinity is associated with increased expression of these proteins [49] . *A. baumannii* resists Quinolones by modulating the antibody binding site where mutation occur in genes encoding the DNA gyrase or one of the targeted topoisomerases , and the bacteria also have flow pumps that cat on the extracellular antagonist tunnel [50] . As for the resistance of *A. baumannii* to aminoglycosides it can be by changing the flow pumps or by expressing the modulating enzymes, especially Acetyltransferases , Nucleotidyl transferases and phosphotransferases . To produce these enzymes , the encode chromosomal or plasmid genes or are paired with integrons and jumpers genes [51,52] . These enzymes modify the hydroxyl or amine group present within these antibiotics and thus reduce the affinity for the target site. Bacteria possess the gene responsible for the 16s rRNA methylation that it prevents significant resistance to most Aminoglycosides such as Amikacin , Tobramycin and Gentamicin [53,54] ,while the mechanisms of bacterial resistance to Tetracycline are modulated the target site on the ribosomal subunit (30s), due to the presence of agene carried on a conjugated plasmide , which encodes for a protein that provides protection the ribosomal subunit of the antibiotic , as well as the possession of agene that encodes then flow systems for this antibiotic [55,56] . As for the mechanism of resistance to polymyxins , it reduced the net negative charge of the outer membrane of the bacteria and thus reduces the affinity between the antibiotic and the surface at the bacteria , as indicated by [57,58] , as the mechanism of action of polymyxins is by stable electrical interactions between the positive charges of polymyxins groups and the negatively charged phosphate groups on Lipid A in lipopolysaccharide (LPS), and resistance can occur as a result in inhibition of certain genes responsible for the biosynthesis of the membrane , of lipid A or the elimination completely[59] . Also [60] indicated that alterations to lipid A with the introduction of phosphor-ethanolamine were present in all isolates of *A. baumannii* anti-colistin resistance.

7.Conclusion

Acinetobacter baumannii considered one of the most important pathogen that cause infectious diseases in hospitals around the world . The emergence of high levels of multi-drug resistant *A. baumannii* made it a priority health issue, especially in hospital and nursing homes for children and the elderly which requires further research and development to discover new antibiotics to reduce deaths from resistant infections around the world. It is also necessary to improve programs to prevent and limit the spread of infection with this pathogens in hospitals.

8.References

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