Acinetobacter baumannii: A Superbug

M. Lowings¹, M.M. Ehlers¹,² and M.M. Kock¹,²

¹ Department of Medical Microbiology, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa
² Department of Medical Microbiology, Tshwane Academic Division, NHLS, Pretoria, South Africa

Acinetobacter baumannii has emerged as a successful opportunistic pathogen, especially in intensive care units (ICU) where A. baumannii infect severely ill patients [1–5]. This micro-organism is known to be a major cause of hospital outbreaks and interinstitutional spread [1, 6–8]. Acinetobacter baumannii is able to survive for extended periods of time in health care settings and can acquire resistance rapidly against antimicrobials [9–11]. Multidrug resistant (MDR) A. baumannii strains are of global concern because these isolates are resistant to at least three classes of antibiotics (carbapenems, cephalosporins, aminoglycosides, fluoroquinolones) and thus limit treatment options [12–14].

Acinetobacter baumannii is widely distributed in health care facilities and can be isolated from medical equipment, bed linen, furniture, medical personnel and indwelling catheters [15, 16]. Acinetobacter baumannii can be transmitted directly from surfaces to patients or through contact with health care workers or it can be transmitted from patient to patient [17]. Risk factors for the acquisition of A. baumannii infections include diabetes mellitus, burn injuries, serious underlying diseases, immunocompromised patients and patients treated with broad-spectrum antibiotics [5, 18, 19]. The clinical manifestations of A. baumannii infections include pneumonia, ventilator associated pneumonia, blood stream infections, meningitis, urinary tract-, respiratory- and wound infections [5, 13, 20, 21]. Only a few virulence factors have been identified in A. baumannii [22–24]. Acinetobacter baumannii has virulence factors associated with invasiveness, transmissibility or the enhanced ability to colonise immunosuppressed patients [15]. Acinetobacter baumannii is capable of forming biofilms, which facilitates colonisation on surfaces and contributes to drug resistance [25]. Jacobs et al. conducted studies with a virulent A. baumannii strain (AB5075) in different animal models [26]. The virulent strain (AB5075) caused more severe infections in the animal models and the survival rates where consistently below 25% [26]. Jones et al. isolated a highly virulent extensively drug-resistant (XDR) A. baumannii strain, even more virulent than the reported AB5075 strain, from an immunocompetent patient [24]. This strain (Clade B isolate) possesses a unique combination of putative virulence genes involved in iron metabolism, protein secretion and glycosylation [24].

Acinetobacter baumannii possesses different antibiotic resistance mechanisms, such as the inactivation of β-lactam antibiotics (penicillins, cephalosporins, carbapenems and monobactams) through the production of extended spectrum β-lactamases, carbapenemases, oxacillinases (OXA) and ampicillin class C (AmpC)-type enzymes [18, 21, 27]. The β-lactamases of Gram-negative bacteria belong to Ambler classes A to D [28, 29]. Broad-spectrum resistance to aminoglycosides (arbekacin, amikacin and gentamicin) is due to ribosomal target-modifying enzymes, which include 16S ribosomal-ribonucleic acid (rRNA) methylases that degrade this specific antibiotic class [5, 21]. Ribosomal target-modifying enzymes include aminoglycoside phosphotransferases, aminoglycoside acetyltransferases and aminoglycoside nucleotidyltransferases [18, 21]. Resistance to quinolones in A. baumannii is associated with: (i) mutations in the gyrA and chromosomal parC genes; (ii) changes in drug entry and efflux and (iii) the quinolone protein, which prevents deoxyribonucleic acid (DNA) to bind to quinolones [30, 31]. Plasmid-mediated quinolone resistance genes encode for DNA gyrase protection proteins [31]. Other resistance mechanisms include: (i) porin deficiency, which is associated with carbapenem resistance due to the reduced antibiotic uptake; (ii) alteration and decreased expression of outer membrane proteins (OMP); (iii) alteration in the affinity of penicillin binding proteins and (iv) overexpression of multidrug efflux pumps that decrease the antibiotic concentration within the cells of A. baumannii [14, 15, 18].

Health care associated infections occur worldwide, with significant economic costs and mortality [32]. Treatment and management of A. baumannii has become a challenge due to the emergence of resistance to antibiotic agents [33–36]. There is a need to further investigate virulence factors and pathogenic mechanisms for more effective control measures in the clinical setting [22, 37].

Keywords Virulence factors; Antimicrobial resistance genes; Resistance mechanisms
Microscopy: advances in scientific research and education

References


[26] Jacobs AC, Thompson MG, Black CC. AB5075, a highly virulent isolate of acinetobacter baumannii, as a model strain for the evaluation of pathogenesis and antimicrobial treatments. 2014;3:e01076-14


