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Editorial

A SLEEPING GIANT AWAKENS: A SILENT THREAT TO GLOBAL HEALTHCARE

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Acinetobacter baumannii has emerged as a formidable yet often underestimated pathogen, earning recognition as a "sleeping giant" and a silent threat to global healthcare systems. Once considered an opportunistic organism of limited clinical relevance, *A. baumannii* has evolved into a leading cause of healthcare-associated infections, particularly in intensive care units, where it is associated with ventilator-associated pneumonia, bloodstream infections, wound infections, and sepsis. Its remarkable ability to survive in hostile hospital environments, rapidly acquire multidrug and extensively drug resistance, and evade host immune responses has significantly complicated treatment and infection control efforts [1].

The name *Acinetobacter* comes from the Latin word for 'motionless' because they lack cilia or flagella to move. Though the genus *Acinetobacter* has 144 species according to recent genomic studies, only *A.baumannii* and *A.lwoffii* have clinical importance, which can be detected by PCR. The recA specific primers are used to detect recA gene in *A.baumannii*, giving a 382 bp fragment. The est specific primers are used to detect est gene in *A.lwoffii*, giving a 309 bp product [2].

Acinetobacter species are typically described as short, plump Gram negative coccobacilli, often appearing in pairs or small chains, which can easily be mistaken for *Neisseria* or *Moraxella*. They are pleomorphic, appearing as rods during rapid growth in the log phase and becoming more spherical/coccoid in the stationary phase [3].

Acinetobacter species are found in soil, water, animals, and humans. *A. baumannii* is a highly adaptable, tenacious pathogen that poses a severe, ongoing threat to healthcare systems globally. Acinetobacter baumannii is a Gram-negative, strictly aerobic, and non-motile coccobacillus that has earned a reputation as a "sleeping giant" or "sleeping demon" or "hidden enemy" in healthcare settings due to its remarkable ability to survive in harsh environments, hospital environments, evade detection, and cause severe, often fatal infections (e.g., ventilator-associated pneumonia, sepsis) when it "awakens" in immunocompromised patients [3].

A. baumannii can enter a Viable But Non-Culturable (VBNC) state. When conditions are harsh, the bacteria "sleep," escaping detection by standard diagnostic methods, and re-awaken to cause relapse in patients. Researchers are focusing on how to detect this pathogen in its dormant state using methods like *PCR. A. baumannii* can persist on inanimate surfaces and lie dormant in hospital environments, surviving on hospital walls, curtains, medical equipment, and surfaces for months, even up to year without food or water, before causing severe, often fatal infections in vulnerable patients. It can withstand dry conditions longer than many other bacterial species [3,4].

Treatment is challenging due to high levels of antibiotic resistance, though carbapenems and polymyxins

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may be used. *A. baumannii* displays a very high multi drug resistance (MDR/XDR/PDR), develops resistance to multiple antibiotic classes, including beta-lactams, aminoglycosides, and quinolones. It is frequently resistant to last-resort antibiotics like carbapenems, creating Carbapenem-Resistant *A. baumannii* (CRAB), with some strains showing Pan-drug resistance (PDR). The World Health Organization (WHO) classifies carbapenem-resistant *A. baumannii* (CRAB) as a top-priority "critical" pathogen, indicating an urgent need for new antibiotics. Proper hygiene and cleaning can reduce the transmission in clinical settings. [5,6].

A. baumannii is a high-priority, multi-drug resistant pathogen utilizing a diverse array of virulence factors for survival and host infection, including capsular polysaccharides (CPS), lipooligosaccharides (LOS), outer membrane proteins (OmpA), and biofilm-associated proteins (Bap). These, along with secretion systems (Type I, II, VI) and iron-acquisition systems (acinetobactin), enable immune evasion, host cell adhesion, and cytotoxic damage [6].

A. baumannii uses a quorum sensing system to regulate virulence, biofilm formation, and antibiotic resistance based on population density, primarily through the abaI/abaR system facilitating survival and enhancing drug resistance. The presence of abaI and abaR is strongly correlated with multi-drug resistance (MDR), especially to carbapenems, in clinical isolates (Figure: 1) [7].

Quorum sensing or quorum signaling is a process of cell to cell chemical communication system used by bacteria to sense population density through the release and detection of signal molecules called autoinducers. Once a critical density is reached, these signals trigger coordinated, community-wide behaviors such as biofilm formation, virulence, and bioluminescence allowing bacteria to act like a multicellular organism. Quorum quenching is a process where agents interfere with, degrade, or block these signals, offering a potential strategy to reduce bacterial virulence without using traditional antibiotics [8].

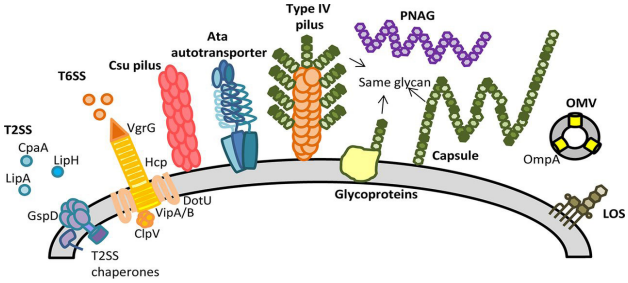

Figure 1: Cell surface components and secretion systems identified in Acinetobacter spp. [9]

A. baumannii (80–91% of strains) form resilient biofilms on abiotic surfaces like catheters and ventilator tubes, protecting it from both disinfectants and the host immune system. It gained notoriety during the Iraq and Afghanistan wars, causing severe, hard-to-treat infections in wounded American soldiers and contaminating military medical facilities during the Iraq War beginning around 2003, and gained a nickname "Iraqibacter" [10].

It is a leading cause of ventilator-associated pneumonia (VAP), bloodstream infections (bacteremia), endocarditis, meningitis, skin, soft tissue and bone infections, wound infections, and urinary tract infections particularly in ICUs. Infections caused by carbapenem-resistant *A. baumannii* (CRAB) have high mortality rates, ranging from 35% to 60%, especially in critically ill, immunocompromised patients. While primarily found in hospitals, it has been isolated from food (raw meat, vegetables) and the environment, suggesting a potential "One Health" problem. It produces carbapenemases, specifically OXA-type (OXA-23, OXA-24/40, OXA-58) and metallo-β-lactamases (NDM, VIM), which break down carbapenems. It possesses efficient efflux pumps, such as AdeABC, that pump antibiotics out of the bacterial cell. It uses outer membrane protein A (OmpA) for adhesion to host cells, and Type IV pili for twitching motility and biofilm establishment. *A. baumannii* uses a capsule to evade phagocytosis and can survive within macrophages [11].

With traditional antibiotics failing, treatment often relies on last-resort drugs like colistin, which is associated with high toxicity (nephrotoxicity). New, promising treatments include cefiderocol, sulbactam-durlobactam (approved in 2023), and eravacycline. Researchers are exploring phage therapy, anti-virulence compounds, and even AI-driven drug discovery (e.g., halicin) to target this pathogen. Phage therapy is being explored as an alternative to antibiotics, often described as a way to "awaken" and eliminate the pathogen [11].

A. baumannii has firmly established itself as a major global healthcare threat, transitioning from a neglected opportunistic pathogen into a leading cause of difficult-to-treat healthcare-associated infections. Its extraordinary capacity for environmental persistence, rapid acquisition of multidrug resistance, and adaptability under selective antimicrobial pressure underscores why it represents a true “sleeping giant” within modern medicine. The convergence of limited therapeutic options, delayed detection, and inconsistent surveillance has allowed *A. baumannii* to spread silently across healthcare

settings worldwide, disproportionately affecting critically ill and vulnerable patient populations [12]. Addressing the growing threat posed by *A. baumannii* requires multifaceted and coordinated global action integrating infection prevention strategies, antimicrobial stewardship, improved diagnostics, and novel therapeutics to prevent this once-silent pathogen from further undermining modern healthcare. Equally critical is the strengthening of global surveillance systems and international collaboration to track resistance trends and guide evidence-based policy decisions.

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