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Tracking *Acinetobacter baumannii* in Critical Care: Environmental Surveillance and One Health Strategies for AMR Prevention

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Abstract

Acinetobacter baumannii (*A. baumannii*) is a bacterium causing infections in hospitals, especially in intensive care units (ICUs). This bacterium persists in hospital environments and quickly becomes resistant to medicines. This review details its behavior, spread, and control in hospital care areas, focusing on environmental monitoring and One Health strategies to stop antimicrobial resistance (AMR). We explain methods to sample and grow bacteria for identification, using quantitative PCR and whole-genome sequencing for rapid detection. We discuss data management frameworks and bioinformatics pipelines that help analyze data and visualize bacterial spread. Control measures include careful cleaning, environmental decontamination machines, and antimicrobial stewardship programs. The One Health perspective emphasizes the connection between humans, animals, and nature in controlling bacteria. Challenges include limited lab space, varied data collection methods, and need for new testing approaches. This report suggests future research in metagenomic surveillance, machine learning-driven risk modeling, and pangenome-guided drug discovery. By integrating laboratory, clinical, and environmental science, this report provides a comprehensive plan to control *A. baumannii* in ICUs and address antimicrobial resistance.

Keywords: *Acinetobacter baumannii,* Environmental surveillance, antimicrobial resistance, One Health, Critical care.

1. INTRODUCTION

A. baumannii is a harmful pathogen increasingly posing a significant challenge in modern hospitals, particularly in critical care environments where severely ill patients are treated. This bacterium, classified as a non-fermentative Gram-negative coccobacillus, does not utilize sugar for nourishment and is shaped like a short rod. It is remarkably resilient, capable of surviving for extended periods on abiotic surfaces such as plastic and metal, including components of ventilators and hospital beds. It can even persist without water for several weeks, facilitating its spread within hospitals, a process known as nosocomial transmission [1,2]. This pathogen is highly adaptable; its genome exhibits plasticity, allowing rapid genetic changes that enhance its resistance to various medications, known as resistance

determinants. Additionally, it can form a biofilm, a sticky layer that enables it to adhere to medical equipment and remain there for prolonged durations.

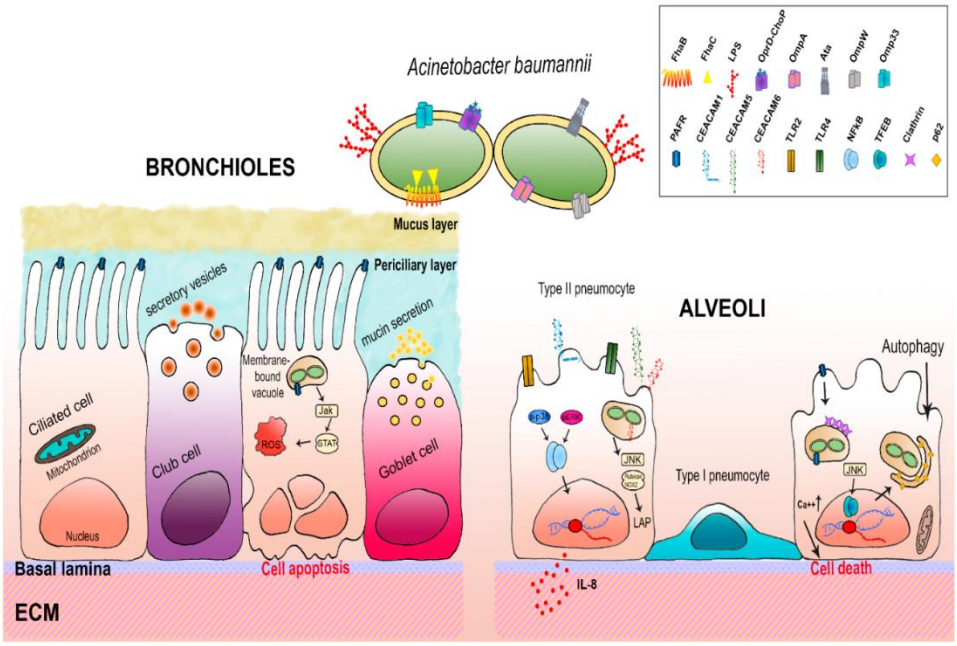

Figure 1: *Acinetobacter baumannii* [3].

Due to these factors, it is challenging to contain this pathogen once it infiltrates environments such as intensive care units (ICUs). Medical professionals encounter significant difficulties in treating patients and preventing the pathogen's dissemination. Therefore, it is imperative to understand the pathogen's survival and proliferation mechanisms within hospital settings. This knowledge will facilitate the development of effective strategies for monitoring and controlling its spread. (See Figure 1).

1.1. Clinical Impact of *A. baumannii* in Critical Care

In ICUs worldwide, *Acinetobacter* poses significant challenges, leading to severe issues such as ventilator-associated pneumonia, bloodstream infections, and wound infections. These infections are extremely dangerous, with mortality rates reaching 40 to 60 out of 100 in certain cohorts [4]. A comprehensive study, involving multiple hospitals reviewing past records, revealed that critically ill patients infected, particularly with the multidrug-resistant *A. baumannii*, were 1.8 times more likely to die compared to those uninfected. This finding held true even after adjusting for the initial severity of illness [5]. Sometimes, individuals carry the bacteria without showing symptoms, a state known as colonization, and may later develop illness. Between 10% and 30% of patients in ICUs are carriers of this microorganism, with the potential to transmit it to others. Between 10% and 30% of patients in ICUs are carriers of this microorganism, with the potential to transmit it to others. [6]. Additionally, patients who fall ill due to this germ tend to have extended ICU stays—about 7 to 14 extra days—heightening the risk of further complications and increasing costs. As a result, this pathogen poses a substantial challenge within hospital settings,

necessitating immediate monitoring and intervention strategies.

1.2. Antimicrobial Resistance (AMR): A Global Threat

Antimicrobial resistance is a growing global concern, as pathogens are increasingly becoming resistant to medical treatments. In 2017, the World Health Organization (WHO) identified Carbapenem-resistant *A. baumannii* (CRAB) as a particularly dangerous strain, necessitating urgent intervention [7]. Data from the Global Antimicrobial Resistance and Use Surveillance System (GLASS) indicate that over 70% of CRAB samples exhibit resistance to carbapenem, a potent antibiotic often considered a last resort for healthcare professionals [8].

In the current scenario, AMR has become a serious concern globally, and is challenging the entire health care system with its increasing resistant mechanisms in antibiotics which we are noticing from last decade. Increasing prevalence of AMR could be because of environmental conditions, geographic location, improper antibiotic usage or misuse of antibiotic prescriptions and protocols [9].

Some bacterial strains are evolving into pan-resistant variants, with only a limited number of potent antibiotics, such as polymyxins or tigecycline, potentially remaining effective. However, these antibiotics are associated with significant toxicity or may exhibit suboptimal pharmacokinetics, indicating that they may not be adequately absorbed or distributed within the body. The scarcity of novel Gram-negative antibiotics in development poses a challenge for treating the most critically ill patients in the future. Addressing this issue necessitates a collaborative approach across human, animal, and environmental health sectors, known as the One Health framework. It is imperative to monitor the dissemination of these bacteria through environmental surveillance, implement antimicrobial stewardship to ensure judicious use of existing medications, and invest in the development of next-generation antimicrobials.

1.3 Objectives of the Review

1. To find out the prevalence and distribution of *A. baumannii* found in important hospitals especially, critical care environments by comprehensive environmental surveillance.
2. To study the molecular mechanisms of *A. baumannii* and its AMR, especially from ICU's.
3. To evaluate the effective the ways of infection prevention and control strategies in preventing *A. baumannii* from spreading in critical care units.
4. To propose and assess combined plans to follow various One Health strategies that try to stop the spread of AMR between people, animals, and the environment.

2. BIOLOGY AND PATHOGENESIS OF *ACINETOBACTER BAUMANNII*

2.1. Taxonomy and General Characteristics

A. baumannii is a Gram-negative, non-fermentative, coccobacillus and grows well in strict aerobic conditions. It belongs to the family Moraxellaceae and order Pseudomonadales. (Table 1).

Scientifically, it belongs to a group of closely related bacteria named as *Acinetobacter calcoaceticus–baumannii complex* (ACB complex). This group also has two other bacteria: *A. pittii* and *A. nosocomialis* [10]. Scientists tell them apart by studying special parts of their genes called 16S rRNA and rpoB gene (16S rRNA and rpoB gene sequence analyses). These bacteria have high genomic similarity but are found in

patients in different amounts and resist medicines in different ways [11].

A. baumannii possesses a relatively small genome (~3.9 Mb) that encompasses numerous genes enabling its survival in diverse environments. These include genes responsible for its outer structures (outer-membrane proteins), mechanisms that expel harmful substances (efflux pumps), and genes capable of relocating within the genome (mobile genetic elements) [12].

Table 1. Taxonomy and general genomic/physiological characteristics of *A. baumannii* [10-12].

Taxonomic Rank	Designation
Domain	Bacteria
Phylum	Proteobacteria
Class	Gammaproteobacteria
Order	Pseudomonadales
Family	Moraxellaceae
Genus	*Acinetobacter*
Species	*A. baumannii*
Genome size	~3.9 Mb
GC content	38–39 mol%
Oxygen requirement	Strict aerobe
Motility	Non-motile

2.2. Virulence Factors

Even though *A. baumannii* does not make strong poisons like some other bacteria lacking classical exotoxins, but presence of virulence determinants makes this organism more harmful.

Important parts include:

- Outer-membrane protein A (Omp A): This helps the bacteria stick to the outside of human cells and can even cause the cells to die by hurting their mitochondria [13].
- Biofilm-associated protein (Bap): This helps the bacteria to form biofilm on things like plastic in hospitals, which helps it survive on surfaces and medical tools [14].
- Phospholipases D and C: These are chemicals that can break the walls of human cells and let the bacteria get inside.
- Metal-acquisition systems (e.g., acinetobactin siderophore): These are special systems that help the bacteria grab iron when there is very little of it in the body [15].

All these parts help the bacteria cause serious infections, especially in very sick patients [13-15].

2.3. Mechanisms of Antibiotic Resistance

The predominant mechanism of carbapenem resistance in *A. baumannii* is attributed to the presence of carbapenem-hydrolyzing enzymes. These include

i. Ambler class A β-lactamases (blaGES-14, blaTEM, blaSHV, blaCTX-M, and blaKPC),

ii. Metallo-β-lactamases (blaIMP-like, blaVIM-like, blaSIM-1, and blaNDM-1), and

iii. Oxacillinases (blaOXA-23-like, blaOXA-24-like, blaOXA-58-like, blaOXA-143, blaOXA-235-like, blaOXA-51-like).

The major expression of OXA genes may be facilitated by Insertion sequence elements (ISs), such as ISAba1, ISAba4, and ISAba125, which provide an additional strong promoter. In addition to resistance, carbapenem-resistant *A. baumannii* (CRAB) also harbors a wide array of virulence factors

that predispose to a worsening course of disease [16].

In the 1970s, scientists identified the global distribution of *A. baumannii*, driven by several lineages known as 'international clones of high risk' (ICs). IC1 and IC2 were first identified, and the presence of these ICs is a major cause of the rapid spread of Acinetobacter and a significant contributor to nosocomial infections in healthcare settings. Environmental and epidemiological surveillance are crucial tools to curtail the spread of this pathogen. Public databases like GenBank currently provide data on the spread and outbreaks of IC1-IC9, which are essential for estimating the risk and global burden of this pathogen. Among these nine ICs, IC2 is the most prevalent clone in the rapid distribution of CRAB [17]. The endemic status of carbapenem-resistant *A. baumannii* in South America has been associated with the production of OXA-23 carbapenemases by international clones IC1 (clonal complex, CC1), IC4 (CC15), IC5 (CC79), and IC7 (CC25). In Argentina, Brazil, Ecuador, Paraguay, Peru, and Venezuela, IC2 (sequence type, ST2) has been recently detected [18].

The biggest danger from *A. baumannii* is that it can fight off many antibiotics. It can do this in many ways.

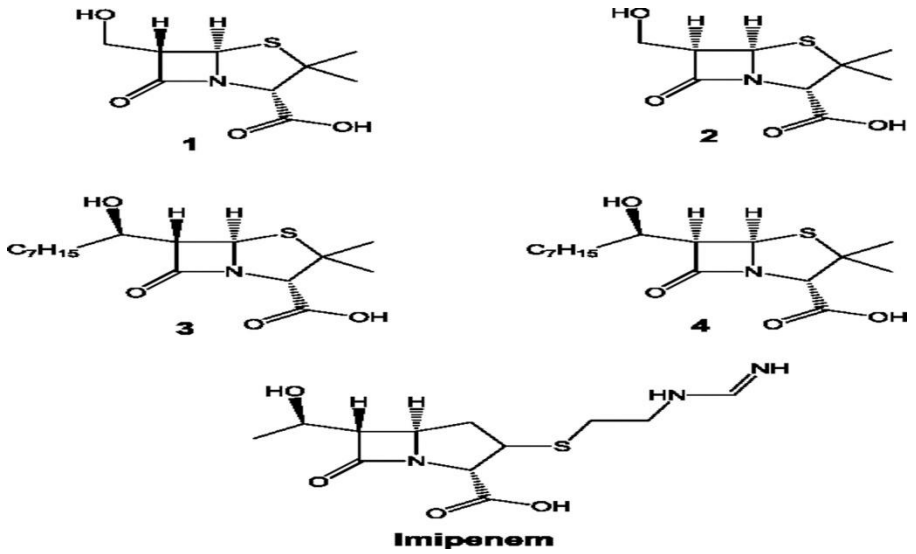

Figure 2: Hydrolytic Mechanism of OXA-58 Enzyme, a Carbapenem-hydrolyzing Class D β-Lactamase from *Acinetobacter baumannii*.

- Intrinsic resistance refers to the inherent ability of bacteria to withstand antimicrobial agents. This resistance is attributed to the presence of an outer membrane that impedes the penetration of drugs and the constitutive production of chromosomal AmpC β-lactamases, enzymes that degrade specific antibiotics [19].
- Acquired resistance mechanisms involve the acquisition of new genetic material, enabling bacteria to counteract novel antimicrobial agents. These mechanisms include
- i) The production of carbapenem-hydrolyzing class D β-lactamases (CHDLs; e.g., OXA-23, OXA-58), which are enzymes that degrade potent antibiotics known as carbapenems,
- ii) Metallo-β-lactamases (e.g., NDM-1), which similarly inactivate antibiotics.

- iii) Additionally, aminoglycoside-modifying enzymes alter aminoglycoside antibiotics, rendering them ineffective [20] (Figure 2).
- The bacteria also employ RND-family efflux pumps (AdeABC, AdeIJK) to expel antimicrobial agents, thereby reducing their efficacy. Mutations in the gyrA and parC genes confer resistance to fluoroquinolones, another class of antibiotics. Furthermore, bacteria can disseminate these resistance determinants to other bacterial cells through horizontal gene transfer mechanisms involving plasmids, integrons, and transposons, which are mobile genetic elements [20,21].
- Another major cause for increasing antimicrobial treatment are integrons, which capture exogenous resistance gene cassettes from different microorganisms and transfer them to others responsible for horizontal gene transfer (HGT), which is an alarming cause of emerging multidrug resistant (MDR) organisms that further leads to the failure of antimicrobial treatment [22].

3. EPIDEMIOLOGY IN CRITICAL CARE SETTINGS

3.1. Incidence and Outbreak Reports

In the last 20 years, *A. baumannii* has become a major cause of nosocomial infections in hospitals and healthcare settings, especially in ICUs. The number of cases is different in different countries. Some places have 5 to 20 cases for every 1,000 days of patient stay in the ICU [23].

Some major outbreaks include:

- Among the 56 *A. baumannii* cases tested for the presence of carbapenemase-resistant genes, 40 cases (71.4%) exhibited the coexistence of both OXA-23 and OXA-51 genes [24]. In a study of 105 CRAB isolates from an intensive care unit in a single hospital in China, collected over six years, all strains were found to carry the blaOXA-23 and blaOXA-66 genes associated with carbapenem resistance. These strains also exhibited a high burden of resistance genes, virulence factors, and insertion sequences. Whole-genome sequencing identified all strains as belonging to ST2, the global clone CC2 [16]. A two-year study conducted in Brazil from 2016 to 2018 revealed that out of 101 Acinetobacter isolates tested, 27 exhibited carbapenem-resistant genes blaOXA-23 and blaOXA-72, which were associated with IC-1, IC-5, and IC-6 [25]. Of 65 isolates, 63 (97%) were resistant to imipenem, consistently associated with acquired carbapenemases, including OXA-23 (80%), OXA-40 (4.6%), OXA-58 (1.5%), or OXA-23/58 (1.5%). Resistance to colistin was observed in 47.7% of the cases [26]. [16,24-26] (Table 2) & (Figure 3).

Table 2. Selected ICU outbreak reports of *A. baumannii* [16,24-26].

S.no	Region	Year(s)	Number of Cases	Carbapenem-resistant genes
1	India	2024	56	OXA-23 and OXA-51.
2	China	2013 - 2018	105	blaOXA-23 and blaOXA-66.
3	Brazil	2016 - 2018	27	blaOXA-23 and blaOXA-72.
4	Southern Europe	2015 - 2018	63	OXA-23, OXA-40 and OXA-58.

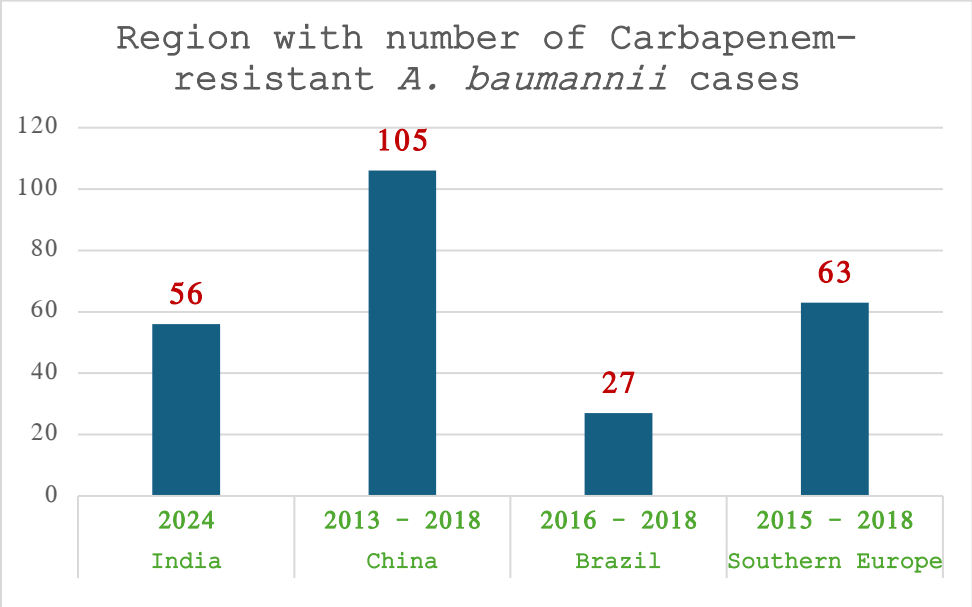

Figure 3: Region with number of Carbapenem-resistant *A. baumannii* cases.

3.2. Transmission Dynamics in Intensive Care Units

The bacteria spread in ICUs because of:

- Places in the hospital with environmental reservoirs,
- Patient colonization,
- And lapses in infection control.

Studies that look at all the DNA of the bacteria genomic epidemiology studies using whole-genome sequencing showed that the same bacteria often spread in the same hospital room. If for example, two bacteria having less than 10 tiny DNA changes (single-nucleotide polymorphism (SNP) distances <10 SNPs) indicates they are from the same outbreak [27].

Doctors' and nurses' hands and dirty hospital tools can also spread vectors, and putting sick patients in the same room helps the bacteria move from one to another [28].

Mathematical modelling shows that cleaning the hospital better could stop up to 30% of the spread, which shows how important cleaning is [29].

3.3. Patient-Related Risk Factors

Some very sick patients are more likely to get infected by *A. baumannii*, especially if:

- They are on Prolonged mechanical ventilation (>7 days),
- They already took broad-spectrum antibiotics, especially carbapenems,
- Or they have undergone any invasive procedures such as central venous catheterization [30].

Patients with long-term diabetes mellitus, chronic obstructive pulmonary disease, or under immunosuppression are also at more risk because their body cannot fight germs well [31].

One large study found that patients with a high acute physiology and chronic health evaluation APACHE II - score (>20), (which is a number that shows how sick a person is), were three times more likely to get CRAB infection. This shows that patients with compromised immune status are more likely to get serious infections [32].

4. ENVIRONMENTAL RESERVOIRS AND SURVIVAL

4.1. Abiotic Surfaces (e.g., medical devices, bed rails)

A. baumannii is a harmful microorganism that can live for a long time on things that are not alive, like Abiotic Surfaces (e.g., medical devices, bed rails). These are things like hospital tools and bed rails that patients touch. This microorganism can stay alive for days on bed rails and even weeks on stainless steel and plastic [6].

The microorganism is capable of forming a biofilm, a cohesive layer that facilitates its adherence to surfaces, including medical devices such as endotracheal tubes and catheters. These devices are employed by healthcare professionals to assist patients with respiration or fluid administration. The biofilm serves to protect the microorganism from desiccation, which refers to the loss of water, and from disinfectants [33], which are chemical agents used to eliminate pathogens. This microorganism exhibits a strong affinity for surfaces composed of polypropylene and polycarbonate, both of which are types of plastic. This adherence is attributed to specific structures known as Csu pili, which are minute, hair-like appendages that aid in attachment, and the BfmRS two-component regulatory system [34], an internal control mechanism that enhances the microorganism's ability to persist and adhere to surfaces (Table 3).

Table 3. Survival of *A. baumannii* on common ICU surfaces and associated adhesion mechanisms [6,33–34]. *PVC – Polyvinyl chloride, PP – Polypropylene.

Surface Type	Survival Duration	Key Adhesion Factors
Stainless steel	≥14 days	Csu pili, Bap
Plastic (*PVC, PP)	≥21 days	Csu pili, OmpA
Glass	~7 days	OmpA, phospholipase D
Medical-grade silicone	≥10 days	BfmRS-regulated biofilm genes

4.2. Water Sources and Plumbing Systems

Moist environments such as sink drains, faucet aerators, and humidifiers serve as potential reservoirs for *A. baumannii.* Research has identified identical strains of *A. baumannii* in respiratory secretions of patients and in sink traps, suggesting that plumbing systems may facilitate the dissemination of this pathogen [35]. In nutrient-limited water systems, *A. baumannii* can enter a viable but non-culturable state, wherein it remains alive but does not proliferate in laboratory cultures. However, it can resuscitate upon entering a more favorable environment, such as a human host [36]. Genetic analyses of these waterborne bacteria frequently reveal biofilm-enhancing gene clusters, which aid in adherence to moist surfaces, indicating niche adaptation [37].

4.3. Role of Healthcare Personnel and Fomites

Healthcare personnel and fomites play a significant role in the transmission of *A. baumannii.* Healthcare workers can transmit pathogens via their hands and through objects such as stethoscopes and mobile phones, which are referred to as mobile fomites. These vectors facilitate cross-transmission of pathogens between individuals. In many hospitals especially in ICUs, hand hygiene compliance rates are below 60%, correlating with increased incidence of *A. baumannii* infections [38]. Swab surveys have

demonstrated that 30% of stethoscopes harbor clonally related strains of *A. baumannii* [39]. Implementation of fomite disinfection protocols and educational interventions has been shown to reduce the spread of *A. baumannii* in ICUs by over 25% [40].

5. ENVIRONMENTAL SURVEILLANCE METHODOLOGIES

5.1. Sampling Strategies and Site Selection

To find *A. baumannii* in hospitals, especially in critical care, we need to follow a good environmental surveillance protocol based on risk-based sampling plans.

To prevent the spread of the microorganisms it is important to screen all high–touch surfaces like bed rail, infusion pumps, ventilator circuits, suction tubing, sink drains, and humidifier reservoirs. [41,42] (Table 4).

How often we test depends on the risk level:

- High-risk places like ventilator tubing should be checked every day or every week.
- Medium-risk places can be checked every two weeks (biweekly).
- Low-risk places (places not touched much) can be checked once a month [43].

Sometimes, people collect microorganisms from many nearby places using composite sampling – pooling. This saves time but might dilute organism recovery. Using individual swabs gives better results, especially for outbreak tracing [44].

Table 4. Risk-based environmental sampling plan for *A. baumannii* surveillance [41-45].

Site Category	Examples	Frequency	Rationale
High-risk surfaces	Ventilator tubing, bed rails, keyboards	Daily/Weekly	Frequent contact; direct patient interface
Water-associated sites	Sink drains, faucet aerators	Weekly	Moist reservoir; VBNC state induces persistence [45]
Medical devices	Catheter hubs, humidifiers	Weekly/Biweekly	Biofilm formation; direct inoculation risk
Low-touch areas	Walls, ceilings	Monthly	Environmental background surveillance

5.2. Culture-Based Detection Techniques

Traditional microbiological techniques remain the cornerstone for isolating microorganisms from environmental samples. To collect samples, researchers employ swabs, which are then inoculated onto sheep blood agar and MacConkey agar and incubated at 35–37 °C for 18 - 24 hours, after thorough examination of colony morphology, Gram stain and oxidase negativity, the isolates are phenotypically confirmed as *Acinetobacter species* and further confirmation is done by using selective media that promote the growth of specific microorganisms i.e. *Acinetobacter*. Examples of such media include Leeds *Acinetobacter* Medium (LAM) and CHROMagar Acinetobacter, which suppress competing flora and enhance the recovery of *A. baumannii* [37,46].

Subsequently, the inoculated plates are incubated at 35–37 °C for 24–48 hours. If *A. baumannii* is present, it will manifest as gray, mucoid colonies. These colonies are verified as *A. baumannii* through oxidase negativity

testing and MALDI-TOF mass spectrometry, a sophisticated technique that identifies microorganisms by analyzing their molecular components [47]. Although culture methods are cost-effective and accessible, they may fail to detect viable but non-culturable (VBNC) cells and are labor-intensive (Table 5).

Table 5. Culture-based detection media for environmental *A. baumannii* surveillance [37,46-47].

Media	Selectivity	Incubation Time	Limitations
Leeds Acinetobacter Medium	High	24–48 h	Does not recover VBNC; false negatives
CHROMagar Acinetobacter	Moderate	24–48 h	May support non-*A. baumannii* species
Mueller-Hinton with imipenem	Low	24 h	Limited specificity; only resistant strains

5.3. qPCR, WGS

Researchers employ specialized molecular assays to accurately and efficiently detect the bacterium *A. baumannii* in environments such as water and surfaces. These assays, which analyze minute biological components like DNA, include quantitative PCR (qPCR). qPCR is a technique used to identify and quantify specific DNA fragments, targeting the blaOXA-51-like gene to confirm the presence of the bacterium. This method is capable of detecting minimal concentrations of the bacterium, as low as 10^3 CFU/mL, within a mere four hours, surpassing traditional culture methods that require bacterial growth and are prone to culture biases [48].

Additionally, multiplex PCR panels, which simultaneously test for multiple targets, can identify resistance genes such as blaOXA-23 and blaNDM-1. These genes confer antibiotic resistance, providing insights into the bacterium's virulence and potential threat [49].

To trace the bacterium's origin and transmission pathways, scientists utilize whole-genome sequencing (WGS), which involves comprehensive DNA analysis. WGS facilitates the identification of single-nucleotide polymorphisms (SNPs), minor alterations in the DNA sequence, and the examination of mobilome structures, which are DNA elements that enhance bacterial adaptability. This approach aids in determining the bacterium's source and dissemination patterns [50] (Table 6).

However, certain limitations exist. The qPCR assay cannot differentiate between viable and non-viable bacteria, and WGS requires high-quality DNA and computational expertise for data interpretation.

Table 6. Comparison of molecular methods for environmental surveillance of *A. baumannii* [48-50].

Technique	Turnaround Time	Sensitivity	Information Yield
qPCR	4–6 h	10^2–10^3 CFU equiv.	Gene-specific detection; quantitation
Multiplex PCR	6–8 h	10^3 CFU equiv.	Resistance gene profiling
WGS	3–5 days	Genome-wide (theoretical)	SNP-level typing; mobilome analysis

5.4. Data Management and Bioinformatics Pipelines

Effective data management is crucial when scientists collect extensive health data, necessitating its organization. This process, known as data management, involves the use of specialized software referred to as laboratory information management systems (LIMS). These systems store critical metadata, including the site of sample collection, the date of collection, and the method employed. Such information facilitates the identification of patterns and enables prompt alerts [51]. For Whole-Genome Sequencing (WGS) data, which provides a comprehensive analysis of an organism's DNA, scientists employ tools such as Null arbor or BioNumerics. These tools assist in quality control, genome assembly, annotation, and SNP calling, which involves identifying small nucleotide polymorphisms [52]. Additionally, dashboards, a type of visual display, can be utilized to further enhance data analysis and interpretation [53]. To ensure interoperability among hospitals or labs, data standardization is employed. This means following the same rules to organize data involves adhering to uniform data organization protocols, such as using the MIxS metadata standards (MIxS stands, which stand sfor "Minimum Information about any (x) Sequence") [54].

6. MOLECULAR TYPING AND OUTBREAK INVESTIGATION

6.1. Pulsed-Field Gel Electrophoresis (PFGE)

PFGE (Pulsed-Field Gel Electrophoresis) is a method used to study bacteria by cutting their DNA into large pieces using special rare-cutting restriction enzymes (like ApaI) [55]. The cut pieces make bands (lines) in a gel, which scientists compare using Dice coefficients (a way to measure how similar the band patterns are). If the similarity is ≥85%, it means the bacteria probably came from the same source (clonal relatedness). PFGE is very good for studying sudden outbreaks, but it takes 4–5 days and it can be difficult to get the same results in different labs because of inter-laboratory reproducibility issues [56].

6.2. Multilocus Sequence Typing (MLST)

MLST (Multilocus Sequence Typing) looks at seven basic genes named as housekeeping genes, like gltA and recA, and gives each group of bacteria a sequence type (ST). This makes it easy to name and share information about bacteria. Scientists can use websites like PubMLST (a public database) to compare bacteria from different places and track how they spread. These are named as clonal complexes, such as CC2 and CC92 [57,58].

Even though MLST is not as detailed as PFGE or WGS, it is very good for long-term tracking and easy to share between scientists [59].

6.3. Whole-Genome Sequencing (WGS) for High-Resolution Tracking

WGS (Whole-Genome Sequencing) looks at all the tiny parts of the DNA, including:

- SNPs (small changes),
- indels (small insertions or deletions),
- and accessory elements (extra pieces of DNA).

Using core-genome SNP analysis, scientists can find out exactly how and where bacteria spread, even if they differ by just 0–5 SNPs [60].

They can also use the computer to do in silico MLST (doing MLST using computer data) and resistome analyses (finding the parts of DNA that make bacteria resistant to antibiotics) all at once [61].

WGS is getting cheaper, but it still needs trained computer experts (bioinformaticians) and agreed rules (standardized pipelines) to get the same results in different places [62].

7. ONE HEALTH FRAMEWORK FOR AMR PREVENTION

7.1. Concept and Relevance to *A. baumannii*

The One Health idea says that human health, animal health, and the environment are all connected. This is important for stopping the spread of AMR [63].

The bacteria *A. baumannii,* which usually affects humans, has also been found in pets, farm animals, and environmental reservoirs [64].

Monitoring all these areas at the same time can help find where the bacteria hide and how they spread. This is important to stop AMR [65].

7.2. Human–Animal–Environment Interface

Farm animals and pets can carry *A. baumannii* bacteria that have carbapenemase genes. These genes are the same as the ones found in sick people, i.e. they can be shared in bidirectional exchange [66]. Wastewater from hospitals can carry resistant bacteria into the environment. There, the bacteria can share resistance genes with others, by horizontal gene transfer [67]. Therefore, it is important to keep an eye on:

- medicine use on farms,
- infections in pets,
- and pollution in water and soil.

This helps stop the spread of very strong bacteria before they come back into hospitals.

7.3. Integrating Veterinary and Environmental Surveillance

To follow the One Health plan, all doctors, vets, and environmental scientists must work together. They should:

- collect samples using the coordinated sampling protocols,
- use shared databases,
- communicate often (cross-sectoral communication channels).

For example, chicken farms could do health checks by taking samples from the birds' bottoms (cloacal swabbing) to test for *A. baumannii*. These samples can be compared with hospital ones to see if they are related [68].

Checking wastewater, irrigation water, and nearby soil helps find where bacteria come, i.e. source attribution and helps make plans to reduce risk (risk modeling) [69]. Groups can then take action together, like using strong biocidal treatments in pipes or using fewer antibiotics.

8. INTERVENTION AND CONTROL STRATEGIES

8.1. Infection Prevention and Control (IPC) Measures

To stop the spread of *A. baumannii* in hospitals, we need good IPC (Infection Prevention and Control) methods. This means:

- Always cleaning hands with alcohol rubs or chlorhexidine soap,
- Patient cohorting or isolation
- Following contact precautions [70]

Doctors and nurses also test high-risk patients early, especially those on prolonged mechanical ventilation. If they are carrying the bacteria, they are kept separate [71]. Also, regular checks and giving feedback to hospital workers help them follow the rules better. One study showed a 40% reduction in ICU infections after using a feedback program [72] (Table 7).

Table 7. Summary of key IPC measures against *A. baumannii* in ICUs [70–72].

IPC Measure	Description	Evidence of Efficacy
Hand Hygiene	Alcohol-based rubs or chlorhexidine soap before/after patient contact	30–50% reduction in transmission rates [70]
Contact Precautions	Gloves and gowns for all patient interactions	35% fewer new colonization's [71]
Patient Cohorting/Isolation	Dedicated rooms or areas for colonized/infected patients	Reduced cross-transmission by 45% [72]
Active Surveillance Cultures	Regular screening of high-risk patients	Enabled early cohorting; 20% fewer outbreaks [71]

8.2. Environmental Decontamination Protocols

Rigorous cleaning of the environment is very important to remove *A. baumannii* biofilms and hidden sources of microorganisms that stay for a long time. (*A. baumannii* biofilms are layers of sticky germs that stick to surfaces and are very hard to clean off.) Cleaning by hand using special cleaning liquids approved by the EPA-approved quaternary ammonium compounds or sodium hypochlorite at 1,000–5,000 ppm chlorine can clean surfaces well. (EPA-approved quaternary ammonium compounds are strong cleaners allowed by the Environmental Protection Agency. Sodium hypochlorite at 1,000–5,000 ppm chlorine means a type of bleach with 1,000 to 5,000 parts of chlorine in every million parts of water.)

But some microbes that are inside biofilms might survive even after this cleaning [73]. Machines can also help clean rooms. These machines use things like hydrogen peroxide vapor (HPV) and UV-C light to kill microorganisms in the air and on surfaces. HPV is a gas made from a cleaning liquid and UV-C light is a special kind of light kills microorganisms in the air and on surfaces. Studies have shown that these machines can lower the number of microorganisms in the environment by 70–90% and also reduce infections in the ICU by 30%. [74,75]. Using HPV after cleaning by hand has worked really well in stopping the spread of microbes when many people get sick at the same time. [76] (Table 8).

Table 8. Comparison of environmental decontamination protocols for ICU settings [73-75].

Method	Active Agent/Mechanism	Reduction in Surface Burden	Considerations
Quaternary Ammonium Compounds	Disrupts cell membranes	2–3 $\log_{10}$ reduction	Cheap, but variable against biofilms [73]
Sodium Hypochlorite (Bleach)	Oxidation of cellular components	3–4 $\log_{10}$ reduction	Corrosive; requires fresh preparation [73]
Hydrogen Peroxide Vapor (HPV)	Reactive oxygen species	4–5 $\log_{10}$ reduction	Automated; cost and downtime considerations [74]
UV-C Irradiation	DNA photodamage	3–4 $\log_{10}$ reduction	Line-of-sight limitation; safety measures [75]

8.3. Antimicrobial Stewardship in Critical Care

Antimicrobial stewardship programs (ASPs) are special plans that help doctors use antibiotics in the best way. The goal is to stop microorganisms from becoming resistant.

Important steps in these programs include:

- Prospective audit with feedback on antibiotic prescriptions.
- De-escalation based on culture results.
- Pre-authorization for restricted agents such as carbapenems and polymyxins [77].

When a hospital used an ASP in a tertiary ICU, the use of carbapenems went down by 25%. At the same time, there was a 15% drop in CRAB incidence [78]. Also, when they used rapid diagnostic stewardship (a fast way to check if someone needs antibiotics), with help from procalcitonin-guided algorithms [79] they were able to cut down the number of days patients took strong antibiotics by 20%. This was done without compromising patient outcomes. (Table 9).

Table 9. Key antimicrobial stewardship strategies and measured impacts in ICUs [77-79].

Stewardship Intervention	Action	Outcome
Prospective Audit & Feedback	Daily review of antibiotic orders	25% ↓ carbapenem use; 15% ↓ CRAB incidence [78]
De-escalation Protocols	Switch to narrower-spectrum agents upon culture results	20% ↓ broad-spectrum days [79]
Pre-authorization Requirements	Restrict use of high-risk antibiotics	30% ↓ inappropriate prescriptions [77]
Rapid Diagnostic Algorithms	Use biomarkers (e.g., procalcitonin) to guide therapy duration	20% ↓ antibiotic exposure days [79]

9. CHALLENGES, GAPS, AND FUTURE DIRECTIONS

9.1. Technical and Logistical Barriers

It is hard to fully watch and control the spread of microorganisms because of many problems. Some places due to limited laboratory capacity or high costs of automated decontamination systems and having shortages of trained personnel [80]. Also, a lack of standardized sampling protocols or data-sharing frameworks, make it hard to compare results [81]. To address this issue, we need to invest in infrastructure and ensure the harmonization of methods so that everyone employs the same techniques.

9.2. Emerging Technologies (e.g., metagenomics, biosensors)

New tools like metagenomics help find microorganisms in the environment without growing them in labs. This can give early warnings. Portable nanopore sequencers are small machines that read DNA quickly right at the hospital, but sometimes make mistakes. So, we need bioinformatics correction to fix the errors [82]. Biosensors, like electrochemical assays targeting OXA-type carbapenemases, can give fast results in less than 1 hour, but they need more testing in real places [83].

9.3. Policy, Workshops, Training, and Capacity Building

To stop AMR for a long time, strong supportive policy frameworks are needed. Governance is needed to mandate environmental surveillance, enforce stewardship guidelines, and help to incentivize development of novel antimicrobials and diagnostics [84]. Training programs for doctors and cleaners in hospitals are very important to ensure that IPC practices are followed to reduce patients' hospital length of stay and to implement strict decontamination practices. Special training centers can support low-resource settings [85].

Collaborative workshops with the WHO should be organized, that emphasize the importance and need to develop advanced curricula and effective training programs on improving antibiotic prescribing practices. Focus on escalating and deescalating the usage of antibiotics when required as per patients' health condition is an important initiative to reduce the impact of AMR [86].

10. CONCLUSION

In this paper, we examined the multifaceted role of the *A. baumannii* pathogen in hospital settings. We discussed its taxonomy, biological characteristics, genomic plasticity, and its ability to adhere to surfaces and cause disease through biofilm formation and a virulence arsenal. This bacterium can survive in arid, nutrient-poor environments. We observed its prevalence in causing illnesses such as ventilator-associated pneumonia and bloodstream infections in hospital ICUs. It is capable of transmission between patients and healthcare workers. We noted its persistence on abiotic surfaces like bed rails and sinks, and its spread via fomites and healthcare personnel.

We compared traditional and modern methods for identifying the pathogen. Traditional methods involve culture-based techniques, whereas modern approaches utilize rapid DNA technologies such as qPCR and whole-genome sequencing (WGS). These advanced tools provide extensive information, necessitating efficient data management pipelines. We found that genotyping methods such as PFGE, MLST, and WGS are instrumental in tracing the origins of outbreaks, with WGS offering the highest resolution.

To mitigate the pathogen, a multifaceted approach is required, including rigorous environmental decontamination, antimicrobial

stewardship, and adherence to infection prevention and control (IPC) protocols. We presented case studies and One Health initiatives where interdisciplinary collaboration successfully curtailed the pathogen in humans, animals, and the environment. However, challenges persist, particularly in resource-limited settings, due to a lack of tools, strategic plans, and trained personnel, underscoring the need for enhanced support and collaboration.

Future Research Prospects

Future efforts to control *A. baumannii* in hospitals will increasingly rely on advanced computational methods and systems thinking. Metagenomic surveillance of environmental pathogens can facilitate early detection of novel pathogens. However, robust pipelines are essential to differentiate between benign and pathogenic organisms and identify genuine threats. Machine learning may aid in predicting future outbreaks by integrating patient data, laboratory results, and safety audits, enabling preemptive interventions.

The discovery of new therapeutics through bioinformatics-driven drug discovery pipelines can be expedited by analyzing *A. baumannii* pangenomes to identify critical targets. Additionally, novel antimicrobial strategies, such as phage therapy and anti-virulence strategies—targeting biofilm-associated regulators or siderophore uptake systems—warrant further investigation through in silico screening and in vitro validation.

Integrating pathogen genomic data with clinical outcomes may elucidate the most virulent strains and ineffective treatments. Emerging real-time, point-of-care sequencing platforms, such as nanopore technology, may soon enable clinicians to identify pathogens at the bedside within hours. However, it is imperative to address accuracy issues and ensure their efficacy in clinical settings. Portable biosensors capable of rapidly detecting virulence factors could also facilitate prompt clinical interventions.

Studying microorganisms in the environment (like water, farms, and sewage) using the One Health will help us learn how they spread outside hospitals. Then, we can make better rules to stop microorganisms from growing and spreading.

In short, to fight this bacterium effectively, a multi-disciplinary approach between computer scientists, doctors, nature experts, and rule makers must work together. With shared information and teamwork, we can reduce *A. baumannii* from spreading between people, animals, and the environment.

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تتبع بكتيريا الراكدة البومانية (أساينيتوباكتر) في الرعاية الحرجة: المراقبة البيئية واستراتيجيات الصحة الواحدة للوقاية من مقاومة مضادات الميكروبات

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الملخص

تعد بكتيريا الراكدة البومانية (*Acinetobacter baumannii*) من المسببات المرضية التي تسبب عدوى في المستشفيات، خصوصًا في وحدات العناية المركزة (ICUs) . تتميز هذه البكتيريا بقدرتها على البقاء لفترات طويلة في بيئات المستشفى، كما تكتسب مقاومة للأدوية بسرعة. يستعرض هذا المقال سلوكها وانتشارها وطرق السيطرة عليها في بيئات الرعاية الصحية، مع التركيز على المراقبة البيئية واستراتيجيات "الصحة الواحدة" للحد من مقاومة مضادات الميكروبات(AMR) . نوضّح طرق جمع العينات وزراعة البكتيريا للتعرف عليها، واستخدام تقنية PCR الكمي وتسلسل الجينوم الكامل للكشف السريع عنها. كما نناقش أُطر إدارة البيانات ومسارات المعلوماتية الحيوية التي تساعد في تحليل البيانات ورسم خرائط انتشار البكتيريا. تشمل إجراءات السيطرة التنظيف الدقيق، أجهزة التعقيم البيئي، وبرامج ترشيد استخدام المضادات الحيوية. تُبرز مقاربة "الصحة الواحدة" الترابط بين الإنسان والحيوان والبيئة في السيطرة على هذه البكتيريا. وتشمل التحديات محدودية المساحات المختبرية، اختلاف طرق جمع البيانات، والحاجة إلى أساليب اختبار جديدة. يقترح هذا التقرير مجالات بحث مستقبلية مثل المراقبة الميتاجينومية، النمذجة التنبؤية المعتمدة على تعلم الآلة، واكتشاف الأدوية الموجهة بواسطة البانجينوم. من خلال دمج العلوم المخبرية والسريرية والبيئية، يقدم هذا التقرير خطة شاملة للسيطرة على *A. baumannii* في وحدات العناية المركزة ومعالجة مقاومة مضادات الميكروبات.

الكلمات الدالة: بكتيريا الراكدة البومانية (Acinetobacter baumannii) ، المراقبة البيئية، مقاومة مضادات الميكروبات، الصحة الواحدة، الرعاية الحرجة