Preprints.org

Article

Not peer-reviewed version

Antibiotic Resistance of *Acinetobacter* Isolated in a Spanish Veterinary Teaching Hospital

Carlota Martínez-Torrecilla , Marta E. García , Marta Pérez-Sancho , Laura Torre-Fuentes , Marta Hernández , María Ugarte-Ruiz , Julio Álvarez * , Jose L. Blanco *

Posted Date: 25 March 2026

doi: 10.20944/preprints202603.2022.v1

Keywords: *Acinetobacter*; antibiotic; animals; veterinary; antimicrobial resistance; gene; isolate; multidrug resistance

Preprints.org is a free multidisciplinary platform providing preprint service that is dedicated to making early versions of research outputs permanently available and citable. Preprints posted at Preprints.org appear in Web of Science, Crossref, Google Scholar, Scilit, Europe PMC.

Copyright: This open access article is published under a Creative Commons CC BY 4.0 license, which permit the free download, distribution, and reuse, provided that the author and preprint are cited in any reuse.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

Disclaimer/Publisher's Note: The statements, opinions, and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions, or products referred to in the content.

Article

Antibiotic Resistance of *Acinetobacter* Isolated in a Spanish Veterinary Teaching Hospital

Carlota Martínez-Torrecilla [1], Marta E. García [1,2], Marta Pérez-Sancho [1,2,3], Laura Torre-Fuentes [3], Marta Hernández [4], María Ugarte-Ruiz [3], Julio Álvarez [1,3,*] and Jose L. Blanco [1,2,*]

[1] Department of Animal Health, Faculty of Veterinary Medicine, Complutense University of Madrid, 28040 Madrid, Spain

[2] Veterinary Teaching Hospital, Complutense University of Madrid, Madrid, Spain

[3] VISAVET Health Surveillance Centre, Complutense University of Madrid, Madrid, Spain

[4] Faculty of Human Medicine, University of Valladolid, Valladolid, Spain.

* Correspondence: jalvarez@ucm.es (J.A.); jlblanco@ucm.es (J.L.B.)

Simple Summary

Acinetobacter is a bacterial genus associated with infections in hospitalised patients. However, its current importance in veterinary centres is unclear. Pet owners are taking the health of their animals more seriously and demand higher standards of care and safety in veterinary clinics and hospitals. Consequently, monitoring the presence of certain bacteria in these centres has a clear relevance for animal health. This study aimed to determine the presence of *Acinetobacter* in a veterinary teaching hospital in Spain. Bacterial isolates were identified using four different identification methods, and two tests were performed to assess antimicrobial susceptibility. The results indicated a low isolation frequency and a low level of antimicrobial resistance of *Acinetobacter*. Nevertheless, as animals can act as reservoirs of *Acinetobacter*, continuous monitoring of this type of bacteria is recommended.

Abstract

Acinetobacter is one of the most relevant pathogenic and nosocomial bacterial genus in human medicine. However, in veterinary medicine, and especially in Spain, there are very few studies about the impact and frequency of infections due to this genus. The main objective of this study was to characterise *Acinetobacter* isolates analysed at the Complutense Veterinary Teaching Hospital of the Complutense University of Madrid, with special emphasis on detecting antimicrobial resistance. A total of 23 isolates obtained from different animal species and samples over a 27-year period, were included in the study. Identification was made by using MALDITOF, VITEK-2, whole-genome sequencing and a chromogenic medium. Antimicrobial susceptibility was interpreted according to CLSI guidelines using the Kirby-Bauer disk diffusion and broth microdilution method. The proportion of clinical isolates identified as *Acinetobacter* spp. at the HCV-UCM was 0.3%. The frequency of AMR was low, though 30.8% of the isolates were classified as multidrug-resistant. Two isolates with the highest MIC for temocillin carried the *tet(X)* gene, and two isolates had mutations in both *gyrA* and *parC* QRDR regions. The results of this study suggest that, in Spain, antimicrobial resistance in *Acinetobacter* isolates of veterinary origin might not be yet widespread.

Keywords: *Acinetobacter*; antibiotic; animals; veterinary; antimicrobial resistance; gene; isolate; multidrug resistance

1. Introduction

Acinetobacter spp. are Gram-negative bacilli or coccobacilli bacteria belonging to the Moraxellaceae family [1], which currently includes 17 genospecies. *A. baumannii* is the most clinically significant species [2].

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

These bacteria are ubiquitous and can be found in soil, water, sediment and as commensals in healthy humans and animals [3], with up to 40% of healthy adults being colonized with *A. baumannii* [4].

A. baumannii is considerate a low-risk pathogen with limited virulence [2] and it is part of the skin and upper respiratory tract microbiota in healthy individuals. However, expression of certain factors such as pili, outer-membrane porins, phospholipases, proteases, lipopolysaccharides, capsular polysaccharides, protein secretion systems, iron-chelating systems, penicillin-binding proteins, and PER-1 β-lactamases can increase its pathogenicity [5]. Furthermore, these species are able to form biofilms in biotic and abiotic surfaces, allowing it to persist in hospital environments. The bacterium remains metabolically inactive in the deepest layers to survive [6] and a positive correlation between biofilm formation and antibiotic resistance has been reported [4].

Other interesting *Acinetobacter* species of increasing clinical relevance are *A. pittii* and *A. nosocomialis* [7,8].

In human medicine, nosocomial infections caused by *A. baumannii* have increased over the past 20 years. This pathogen is the main responsible of sepsis, pneumonia, meningitis, and endocarditis in intensive care and burn units, after methicillin-resistant *S. aureus* (MRSA) [8]. In hospital environments, transmission can occur through people and through fomites (reusable medical equipment, pillows, sheets, gloves, etc.) [2]. Risk factors for infection include the use of venous and urinary catheters, long periods in hospitalization, and recent surgeries [4]. Mortality in vulnerable patients can reach 60% due to lack of effective treatments in cases of pneumonia and bacteraemia. The treatment of choice for these pneumonias are carbapenems combined with other antibiotics, for example, nebulized colistin [9].

In veterinary medicine, studies about frequency of infections and clinical significance of *Acinetobacter* spp. are scarce, although *A. baumannii* is considered an emerging pathogen [4] and has been isolated from multitude of animal species [10]. In animals, a high mortality rate (70%) has been described, similar to human medicine [11].

The situation in companion animals (mainly hospitalized dogs and cats) is the most concerning one, owing to the isolation of the same *Acinetobacter* strain in pet and owner. This suggest that pets may act as reservoirs for humans because of their closeness [3,12]. For instance, a study of two *A. baumannii* outbreaks in a veterinary hospital in the Netherlands found that both clusters were related to RUH-134 (the human European clone II reference strain), and a SNP-based analysis suggested that the ancestors of the two outbreaks appeared in the 80's. [10]. The risk factors, in this case, were hospitalization and antibiotic treatment [6].

The European Committee on Antimicrobial Susceptibility Testing (EUCAST) indicates that *Acinetobacter* genus is naturally resistant to the following antibiotics: ampicillin, amoxicillin, amoxicillin-clavulanic acid, cefazolin, cefotaxime, ceftriaxone, aztreonam, ertapenem, trimethoprim, tetracycline, fosfomycin and tigecycline [13]. The antibiotic groups that are active against this genus are carbapenems, polymyxins, fluoroquinolones and aminoglycosides.

Nowadays, carbapenems are the antibiotics of choice to treat infections due to *A. baumannii*, but carbapenem-resistant strains have already been described. Carbapenem-resistant *A. baumannii* has been classified as "Critical Group" by the World Health Organization (WHO) [14]. In addition, strains resistant to all antibiotic groups have been also described, including β-lactams, aminoglycosides, cephalosporins, carbapenems, fluoroquinolones, trimethoprim/sulfamethoxazole, and recently colistin (though the mechanisms in not fully studied yet) [8].

For instance, a study from a Veterinary Clinical Hospital in South Africa found that 95% of isolates were resistant to at least one antibiotic, and 60% were classified as multidrug-resistant (MDR) [15].

According to the "One Health" concept, health of humans, animals and ecosystems are closely interrelated.

Therefore, any changes in these relationships may increase the risk of appearance and propagate new diseases at the human-animal interface. For this reason, it is of paramount importance to

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

characterize the dynamics of bacterial species shared between animals and humans in the veterinary field. Around 60% of emergence infectious diseases are originated from animals [16]. In addition, there is a lack of studies on *Acinetobacter* spp. in veterinary medicine, particularly in our country.

Because of all this, we performed an investigation to assess the epidemiological situation and the relative frequency of isolation of *Acinetobacter* spp. infection in animals visiting the Veterinary Clinical Hospital of the Complutense University of Madrid (HCV-UCM). Moreover, we studied antimicrobial resistance, focusing on finding possible multidrug-resistant bacterial strains.

2. Materials and Methods

All isolates previously identified as *Acinetobacter* spp. according to VITEK-2 and stored in the Microbiology and Parasitology Laboratory at the HCV-UCM, were considered in this study. Initially, 27 isolates presumptively identified as *Acinetobacter* spp. were identified out of a total of 4.560 isolates, with the first isolate found in 2000 and the last in 2024. Only 23 were successfully recovered in blood agar and included in the present study. Table 1 provides detailed information of these 23 isolates.

Table 1. Strain identification, specie, date of primo isolation, and sample type of the 23 isolates.

Strain Identification	Specie	Date of primo isolation	Sample
1125	Equid	14/09/2009	Corneal cotton swab
1156	Equid	22/10/2009	Skin cotton swab
1409	Canine	31/05/2010	Otic exudate
1308	Tortoise	17/01/2011	Tracheal exudate
1643	Equid	29/11/2014	Abdomen incision
1963	Equid	05/05/2016	Uterine fluid
2080	Ovine	25/10/2016	Soft tissue
2724	Canine	26/09/2018	Skin and hair
3321	Galapagos tortoise	17/11/2020	Exudate
3386	Feline	02/03/2021	Soft tissue
3534	Feline	28/06/2021	Urine
3556	Mouse	12/07/2021	Ocular cotton swab
3627	Feline	11/10/2021	Tracheal lavage
3684	Tortoise	03/11/2021	Shell Wound cotton swab
3815	Feline	11/02/2022	Tracheal lavage
3904	Canine	24/03/2022	Wound exudate
4062	Rabbit	14/09/2022	Nasal sample
4070	Feline	22/09/2022	Tracheal lavage
4255	Bovine	22/12/2022	Cerebrospinal fluid
4338	Cockatoo	13/03/2023	Lymph
4348	Canine	13/03/2023	Wound and abscess
4485	Chameleon	12/06/2023	Conjunctive exudate

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

4676	Canine	25/09/2024	Otic exudate

Successfully recultured isolates were subjected to bacterial identification using MALDI-TOF and WGS to recognise the bacterial genus and specie. Manufacturer-recommended identification scores for MALDI-TOF were followed. However, only scores higher than 2.000 were considered for assigning *Acinetobacter* species. In addition, cultures were grown on CHROMagar™ Acinetobacter, a specific and differential chromogenic medium for *Acinetobacter* species.

Antimicrobial susceptibility testing was performed using two methods, the Kirby-Bauer disk diffusion method and the broth microdilution method. The Kirby-Bauer method was performed and interpreted according to the Clinical & Laboratory Standards Institute (CLSI), specifically, the breakpoints used are presented in the CLSI vs FDA breakpoints comparison document [17].

Briefly, the Kirby-Bauer disk diffusion method was made on Mueller-Hinton agar, with six antibiotics tested per plate. A total of 24 antibiotics from different pharmacological groups were evaluated: amoxicillin-clavulanic acid (OXOID; 3µg), amikacin (OXOID; 30µg), ampicillin-sulbactam (OXOID; 20µg), cefotaxime (BIO-RAD; 30 µg), cefepime (BIO-RAD; 30 µg), cefotixin (BIO-RAD; 30 µg), ceftazidime (BIO-RAD; 30 µg), clindamycin (OXOID; 10µg), chloramphenicol (OXOID; 30µg), erythromycin (OXOID; 10µg), gentamicin (OXOID; 10µg), impinem (OXOID; 10µg), levofloxacin (OXOID; 5µg), meropenem (OXOID; 10µg), oxacillin (OXOID; 1µg) , polymyxin B (OXOID; 300iu), rifampicin (OXOID; 5µg), temocilin (OXOID; 30µg), tetracycline (OXOID; 30µg), tigecycline, and vancomycin (OXOID; 5µg). These antibiotics were selected based on the literature, antibiotics most frequently used according to the HCVC protocol, and those included in prior related studies.

The broth microdilution method was performed according to ISO 20776-1:2019 guidelines. Minimum inhibitory concentrations (MICs) for amikacin (AMI), sulfamethoxazole (SMX), trimethoprim (TMP,) ciprofloxacin (CIP), tetracycline (TET), meropenem (MERO), azithromycin (AZI), nalidixic acid (NAL), chloramphenicol (CHL), cefotaxime (FOT), tigecycline (TGC), ceftazidime (TAZ), colistin (COL), ampicillin (AMP) and gentamicin (GEN) were determined in a selection of isolates using the two-fold broth microdilution reference method following commercial panel EUVSEC3 (Trek Diagnostic Systems; Thermo Scientific, Waltham, MA, USA). Interpretation of the quantitative data was carried out according to the guidelines of CLSI [17].

Bacterial genomic DNA was extracted and purified using Qiagen DNA Blood and Tissue Kit, following the manufacturer's instructions, and quantification of the DNA concentration was done using a Qubit® fluorometer (Invitrogen). WGS libraries were prepared with the Nextera XT DNA Library Preparation Kit (Illumina) following manufacturer instructions. The concentrations of each library were adjusted to 4 nM to obtain equimolar DNA concentrations in a single pool of libraries and sequenced in a MiSeq platform (Illumina).

Raw reads obtained from sequencing were filtered out with Trimmomatic [18] and evaluated with FastQC [19]. Reads that passed the quality control were characterized using Kraken [20]. Those reads identified as *Acinetobacter* were assembled with SPAdes [21] and the quality of the assemblies was evaluated with QUAST [22] and CheckM [23]. Assemblies were screened for the presence of antibiotic resistance genes with AMRFinder v3.11.2 with database version 2022-12-19.1 [24].

3. Results

3.1. Acinetobacter Isolation

27 isolates (0.6%) were identified as *Acinetobacter* spp. *Acinetobacter* was isolated from tracheal lavage and exudate samples (5/23 = 21.7%), wound and incision samples (4/23 = 17.4%), ocular samples (3/23 = 13%), skin samples (2/23 = 8.6%), otic exudates (2/23 = 8.6%), uterine fluid (1/23 = 4.3%), urine (1/23 = 4.3%), and cerebrospinal fluid (1/23 = 4.3%).

Among the 23 isolates included in this study, 5/23 were from cats (21.7%), 5/23 were from dogs (21.7%), 4/23 were from horses (17.4%), 3/23 were from tortoises (13%), 1/23 was from a sheep (4.2%),

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

1/23 was from a mouse (4.2%), 1/23 was from a rabbit (4.2%), 1/23 was from a cow (4.2%), and 1/23 was from a chameleon (4.2%).

3.2. Acinetobacter Identification

Table 2 shows the comparison of the results obtained using the four identification methods applied in the study: MALDI-TOF, VITEK-2, whole genome sequencing (WGS), and chromogenic medium. MALDI-TOF, VITEK-2 and WGS can identify isolates at the species level, whereas the chromogenic medium provides a qualitative assessment of the possible growth of *Acinetobacter* spp.

Table 2. Identification results of 23 isolates obtained using MALDITOF, VITEK-2, WGS and chromogenic medium.

I.D of the isolates	WGS	MALDI-TOF	VITEK-2	[1]CHROMOGENIC MEDIUM
1125	*A. pitti*	*Acinetobacter* spp.	*Acinetobacter* spp.	+
1156	No identification	[2]No identification	*A. iwoffii*	+
1308	No identification	*Alcaligenes faecalis*	*A. iwoffii*	+
1409	*A. baumannii*	*A. baumannii*	*Acinetobacter* spp.	+
1643	No identification	No identification	*Acinetobacter* spp.	+
1963	*A. baumannii*	*A. baumannii*	*A. baumannii*	+
2080	*A. indicus*	No identification	*A. iwoffii*	+
2724	*Moraxella catarrhalis*	*Moraxella canis*	*Acinetobacter* spp.	-
3321	*A. bereziniae*	*Acinetobacter* spp.	*A. iwoffii*	+
3386	*A. radioresistens*	*A. radioresistens*	*A. radioresistens*	+
3534	*Solibacillus silvestris*	*Solibacillus silvestris*	*A. iwoffii*	+
3556	*A. baumannii*	*A. baumannii*	*A. baumannii*	+
3627	*Neisseria* spp.	*Neisseria flavescens subflava*	*A. iwoffii*	-
3684	*Acinetobacter* spp	*Acinetobacter* spp	*A. iwoffii*	-
3815	*Neisseria* spp.	No identification	*A. iwoffii*	-
3904	*A. calcoaceticus*	*Acinetobacter* spp	*A. baumannii*	+
4062	*Staphylococcus hominis*	*Staphylococcus hominis*	*Acinetobacter* spp.	+

[1] The "+" sign indicates the growth of pink bacterial colonies, which is related to a possible *Acinetobacter* species, and the "-" sign indicates the growth of blue bacterial colonies, suggestive of the growth of something differente to *Acinetobacter*.

[2] "No identification" is usually associated wiht the abscence of isolate information in the MALDI-TOF database, or with a very differente bacterial spectrum.

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

4070	*Glasserella parasuis*	*Staphylococcus hominis*	*A. iwoffii*	-
4255	*A. iwoffii*	*A. pseudolwoffii*	*A. iwoffii*	+
4338	*A. baumannii*	*A. baumannii*	*A. baumannii*	+
4348	*A. baumannii*	*A. baumannii*	*A. baumannii*	+
4485	*A. bereziniae*	*Acinetobacter* spp	*A. iwoffii*	+
4676	*Moraxella catarrhalis*	*Moraxella canis*	*A. iwoffii*	+

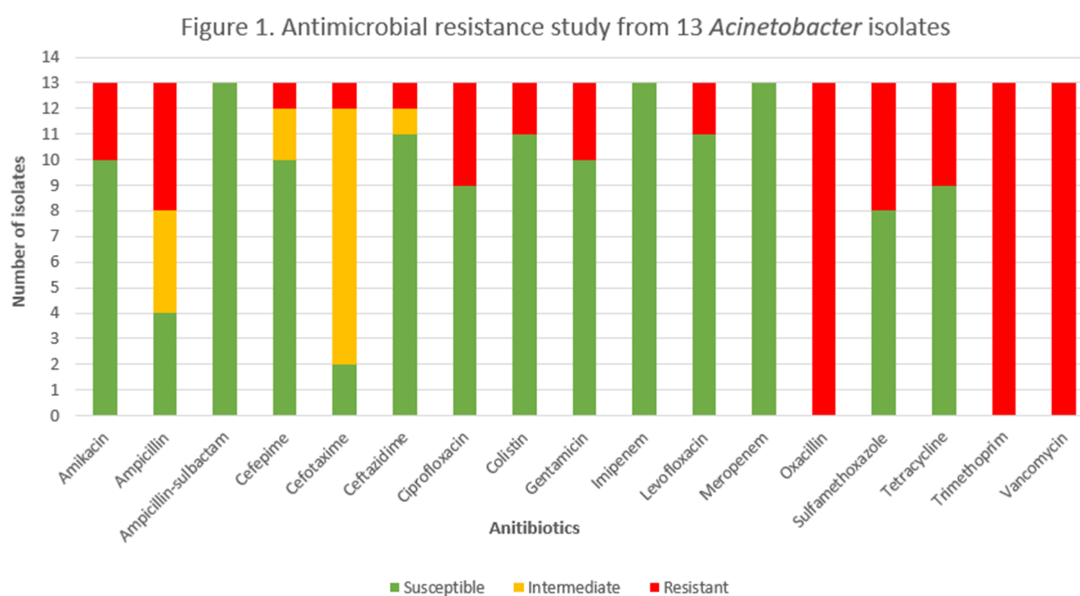
According to the WGS results, 13 (56.5%) were confirmed as *Acinetobacter* spp. 5 out of 13 belong to the *A. baumannii* species (Table 2). MALDI-TOF identified as part of the *Acinetobacter* genus 12 (52.2%) of the 23 isolates, almost half of which would be *A. baumannii* (5/23). VITEK-2 identified the bacterial growth as *Acinetobacter* spp. in 100% of the isolates, and 21.7% (5/23) as *A. baumannii* species.

Finally, the chromogenic medium found positive growth compatible with *Acinetobacter* spp. in 78.2% of the isolates (18/23).

MALDI-TOF was the identification method with the highest concordance with WGS, reaching 92.3% of observed agreement at the genus level (12/13) and 46.2% at the species level among the *Acinetobacter* spp. isolates (6/13). Agreement was higher for VITEK-2 when considering identification of isolates as belonging to the *Acinetobacter* genus (100%), but much lower when considering identification of *A. baumannii* (30.8%). In the case of the chromogenic medium, 92.3% of concordance was the value for genus level. With this chromogenic medium we cannot specify species level of *Acinetobacter*, so no data can be interpretable. Results from all four identification methods matched in 38.5% of the isolates identified as *Acinetobacter* spp. by the WGS (5/13).

3.3. Acinetobacter Antibiotic Resistance

The number of isolates with a resistant phenotype for each antibiotic is shown in Figure 1. The number of resistant isolates ranged between 4 and 6, with higher levels of resistance (13/13) for oxacillin, trimethoprim and vancomycin, and lower to carbapenems (full susceptibility).



© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

Figure 1. Antimicrobial resistance study from 13 *Acinetobacter* isolates[3].

As shown in Figure 1, the phenotype for certain antibiotics (amoxicillin-clavulanic acid, azithromycin, cefotixin, clindamycin, chloramphenicol, erythromycin, nalidix acid, polymyxin B, rifampicin, temocillin, tigecycline) could not be interpreted due to the lack of specific breakpoints in the CLSI vs FDA breakpoints comparison document [17] for the *Acinetobacter* genus.

The following mechanisms linked to AMR were identified in the 13 *Acinetobacter* isolates: *amvA, msr(E), mph(E), mef(F), msr(G), nreB, tet(X3), adeC, tet(B), tet(39), sul2, sul1, merE, merD, merA, merT, merR, aph(6)-Id, aph(3")-Ib, aac(6')-Ib4, aph(3')-Vla, ant(3")-IIa, aac(3)-IIe, aac(3)-IVa, ant(2")-Ia, aac(6')-Ian, aac(3)IId, abaF, qacEdelta1, arr-3, blaADC* variants (*blaADC166, blaADC26, blaADC76, blaADC165*) and several *blaOXA* variants (*blaOXA106, blaOXA64, blaOXA144*), *blaCARB-16, blaBRO, dfrA44, parC_S84L, gyrA_S81L, adeS_H189Y,l floR, ars(B), dfrA40*. The 13 *Acinetobacter* isolates carried between 1 and 24 AMR mechanisms (mean =7.3 mechanisms/isolate).

Table 3 shows the antibiotic resistance genes organised according to the associated antibiotic.

Table 3. Antimicrobial resistance mechanism described in 13 *Acinetobacter* isolates[4].

Antibiotic	Antibiotic resistance gen
Amikacin	aph(3')-VIa (1), aac(6')-Ib4 (1), aac(3)-Iva (1) aac(6')-Ian (1), adeS_H189Y (2), aph(6)-Id (5), ant(3")-Iia (5), aph(3")-Ib (4), aac(3)-Iie (1), ant(2")-Ia (1)
Amoxicillin-clavulanic acid	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Ampicillin	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaBRO (0), blaCARB-16 (0), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), adeS_H189Y (0)
Ampicillin-sulbactam	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Azithromycin	msr(E) (2), msr(G) (0), mef(F) (0), mph(E) (2), amvA (7), adeS_H189Y (2)
Cefepime	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Cefotaxime	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Cefotixin	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Ceftazidime	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)

[3] Columns without data correspond to antibiotics which the CLSI guidelines do not have interpretative criteria for.

Isolates were classified as resistant (represented with the letter "R" and red colour), intermediate (letter "I" and yellow) or susceptible (letter "S" and Green) depending on the diameter of the inhibition zone (Kirby-Bauer method) or the established breakpoint (broth microdilution method) for each antibiotic.

[4] The number of isolates carrying each gene is between brackets

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

Ciprofloxacin	gyrA_S81L (2), parC_S84L (2), adeS_H189Y (2)
Clindamycin	—
Chloramphenicol	floR (2), adeS_H189Y (2)
Colistin	—
Erythromycin	msr(E) (2), msr(G) (0), mef(F) (0), mph(E) (2), amvA (7), adeS_H189Y (2)
Gentamicin	aac(3)Iid (1), aph(3')-VIa (1), aac(6')-Ib4 (1), aac(3)-Iva (1) aac(6')-Ian (1), adeS_H189Y (2), aph(6)-Id (5), ant(3")-Iia (5), aph(3")-Ib (4), aac(3)-Iie (1), ant(2")-Ia (1)
Imipinem	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Levofloxacin	gyrA_S81L (2), parC_S84L (2), adeS_H189Y (2)
Meropenem	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Nalidixic Acid	gyrA_S81L (2), parC_S84L (2), adeS_H189Y (2)
Oxacillin	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Polymyxin B	—
Rifampicin	arr-3 (1)
Sulfamethoxazole	sul1 (1), sul 2 (5)
Temocillin	blaADC (2), blaADC26 (2), blaADC76 (1), blaADC165 (1), blaADC166 (1), blaOXA (7), blaOXA64 (2), blaOXA106 (1), blaOXA144 (1), blaBRO (0), blaCARB-16 (0), adeS_H189Y (0)
Tetracycline	tet(B) (2), tet(39) (1), adeS_H189Y (2), adeC (2), tet(X3) (2)
Tigecycline	tet(B) (2), tet(39) (1), adeS_H189Y (2), adeC (2), tet(X3) (2)
Trimethoprim	dfrA40 (0), dfrA44 (1)
Vancomycin	—

Certain genes, such as *blaOXA* and *blaADC* variants, were found in *Acinetobacter* as expected. Other beta-lactamase encoding genes which could have been acquired horizontally (*blaCARB-16*, *blaBRO*) were also found, though no carbapenemase-associated genes were detected. Of note, the two isolates with the highest MIC for temocillin (8 ug/ml and 1 ug/ml compared to 0.5 or <0.25 for the rest) carried the *tet(X)* gene, and two isolates had mutations in both *gyrA* and *parC* QRDR regions (both with a resistance phenotype to CIP and NAL). Besides, from 17 to 24 AMR markers were detected in two isolates (Table A1).

A table providing a more detailed association between each resistance gene/mechanism gene and the phenotypic susceptibility of each strain is presented in the supplementary material (Table S1).

4. Discussion

Out of the 23 isolates initially considered as potential *Acinetobacter*, 13 were confirmed as *Acinetobacter* based on WGS results. The very low proportion of clinical isolates presumptively identified as *Acinetobacter* spp. (0.6%), coupled with the even lower proportion of confirmed *Acinetobacter* spp. isolates (0.3%). This indicates that the clinical relevance of this genus in animals visiting the HCVC is very low, in agreement with a previous study also describing a low frequency

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

(6.5%) of isolation of *Acinetobacter* spp. in dogs and cats in Reunion Island [6]. However, other studies based on syndromic surveillance described higher prevalence levels of *Acinetobacter* spp. infection in dogs (16.3%) and cats (12%) [11].

This suggests that the true incidence of this genus in hospitals and veterinary clinics remains unknown and may even vary depending on the country and type of surveillance applied.

The majority of *Acinetobacter* isolates came from tracheal lavage and tracheal exudates (21.7%), wound and incision samples (17.4%), and ocular samples (13%). These results differ from those reported by Van der Kolk [8]. They observed that, in dogs, *Acinetobacter* was most commonly isolated from pyoderma, chronic eczema, systemic and local infections, vaginal or saliva samples, and urinary infections; whereas, in cats, it was most frequently isolated from intravenous catheters, necrotizing fasciitis, septic shock, or liver biopsies. This diversity of results highlights the great opportunistic capacity of *Acinetobacter*, which can be isolated from any type of sample.

Regarding the animal species which *Acinetobacter* was isolated from, our results are consistent with the literature, with the majority of the isolates coming from companion animals, mainly dogs and cats [10].

Relating to bacterial identification and considering the WGS as the gold standard, MALDI-TOF was the most effective method, with a high reliability to identify isolates as *Acinetobacter* spp. (92.3%). MALDI-TOF was more limited when considering specific *Acinetobacter* species (correctly identified 46.2% out of the 13 isolates), otherwise these values were lower for the species level in VITEK-2. This species-level identification limitation for *Acinetobacter* using MALDI-TOF, has also been reported in other studies [31–33], which concluded that this method is highly reliable for identifying *A. baumannii*, but WGS is recommended for the remaining species. The chromogenic medium could be recommended as a rapid and effective first-line screening tool since it showed a near-complete concordance with WGS (92.3%).

The reason why we have chosen 15 antibiotics for the broth microdilution method (some of them were also included in the disk diffusion test) is because for antibiotics such as ampicillin, colistin, or trimethoprim, CLSI does not provide interpretable values for the disk diffusion method. By combining both methods, a broader spectrum of antibiotics could be evaluated.

Referring to antimicrobial susceptibility results, *Acinetobacter* has intrinsic resistance to multiple antibiotics, including several that are part of the EUVSEC3 plate and disk diffusion test (ampicillin, amoxicillin-clavulanic acid, cefotaxime, trimethoprim, and tetracycline) and therefore their results were not considered to classify the strains as MDR.

Despite these intrinsic resistances, several susceptible and intermediate strains to these antibiotics have been described.

For example, 30.7% (4/13 isolates) were susceptible and 38.5% (5/13 isolates) were intermediate to ampicillin; 15.4% (2/13 isolates) were susceptible and 84.6% (11/13 isolates) were intermediate to cefotaxime; and 69.2% (9/13 isolates) were susceptible to tetracycline. Similar findings have also been reported in other studies, such as those by Benga [3], Hamouda [25], or Sebola [15], in which susceptibility to tetracycline was observed.

All of this creates two question: why certain bacterial strains show antibiotic resistance genes but they are susceptible to those antibiotics? Why are there other bacterial strains without detectable resistance genes that show phenotypic resistance?

All 13 confirmed *Acinetobacter* isolates (100%) exhibited resistance to at least one antibiotic. This result is similar to the Sebola et al. [15] study, in which 95% of the isolates were resistant to at least one antibiotic in their veterinary teaching hospital. In our study, overall, high levels of antibiotic resistance were not observed. More than half of the isolates were susceptible to all antibiotics tested, except for ampicillin, cefotaxime, trimethoprim, oxacillin and vancomycin.

Resistance to aminoglycosides (amikacin and gentamicin), fluoroquinolones (ciprofloxacin, levofloxacin), sulphonamides (sulfamethoxazole), tetracyclines (tetracycline), and polymyxins (colistin) was found in 2-5 isolates. We want to highlight the resistance to vancomycin and colistin.

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

Both are antibiotic for human use. Vancomycin is considered "A category" antibiotic, and colistin is in category B according to the PRAN [26].

Resistance to colistin is especially worrying as it represents a last-resort therapeutic option in human medicine for treating MDR *Acinetobacter* infections.

We considered MDR bacteria as those non-susceptible to at least one antibiotic in three or more pharmacological classes [27]. In our study, we classified 30.8% of isolates as MDR (4/13 isolates). This proportion is lower than previously reported [11,15], but still above the ideal scenario of a prevalence of MDR bacteria close to zero.

While a large variety of AMR mechanisms was detected in one to seven of the 13 sequenced *Acinetobacter* isolates, some of them were expected considering the previous description of carriage of certain genes such as beta-lactamase encoding genes in this genus [28]. Still, finding the *tet(X)* gene, even at a low frequency (2/13 isolates: strain "1125" from 2009 and strain "4255" from 2022), is a more concerning result, particularly given the increased MIC observed especially in one of the two *tet(X3)*-carrying isolates. Previous studies have described the dissemination of *tet(X3)*-carrying plasmids in *Acinetobacter* of animal origin [29], further highlighting the risk of horizontal spread of such MGE in one health settings. Similarly, the presence of QRDR mutations in CIP and NAL-R *A. baumannii* isolates, while also previously found at higher frequency in clinical collections [30] should be also be further considered.

5. Conclusions

Nowadays, health and welfare of companion animals are becoming a more frequent focus of discussion. As a result, owners increasingly demand the highest standard of medical care for their pets and seek veterinary hospitals that offer the greatest possible quality and safety services. For this reason, conducting studies such as the present one in national reference hospitals, where the impact and relevance of certain opportunistic bacteria are currently not well established, is becoming increasingly important.

The results of this study suggest that, in the hospital under study, antimicrobial resistance might not be yet widespread among *Acinetobacter* isolates of veterinary origin. Nevertheless, as animals can act as reservoirs of resistant strains, continuous monitoring of antimicrobial susceptibility by validated methods is recommended.

Finally, we hope that the data generated in this study, including the results obtained in the disk diffusion and broth microdilution method, may contribute to the establishment of new breakpoints for this bacterial genus and for certain antibiotics for which information is still lacking. Furthermore, these data may assist colleagues in finding a better understanding of the true impact of *Acinetobacter* in veterinary medicine.

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org, Table S1: Correlation between phenotypic susceptibility and resistance genes in 13 isolates. .

Author Contributions: Conceptualization, J.L. and M.E.; methodology, J.L. and M.E.; software, L.T.; validation, J.L. and M.E.; formal analysis, J.A., L.T., M.H.; investigation, J.L. and M.E.; resources, C.M; data curation, J.A., L.T., M.U.; writing—original draft preparation, C.M.T.; writing—review and editing, C.M, J.A, L.T., J.L.; supervision, J.L., M.E., M.P.; project administration, M.P.; funding acquisition, J.A. All authors have read and agreed to the published version of the manuscript.

Funding: This research was made under the *PID2021-125136OB-I00 (AMR-EPIPLAS)* funded by MCIN/AEI and ERDF – a way of making Europe. And it is also a contribution to PID2024-1595580B-I00 funded by University, Innovation and Science Ministry (MICINN).

Data Availability Statement: Whole genome sequences are updated at the European Nucleotide Archive web (ENA: https://www.ebi.ac.uk/ena/browser/home).

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

Acknowledgments: We would like to thank the technicians of the Parasitology and Microbiology Laboratory at HCVC for the excellent and well-organized isolation and storage of the strains used in this research. We also like to thanks Estefanía Rivero Muñoz, Nisrin Maasoumi and María García for their valuable contributions to the laboratory analyses at VISAVET Health Surveillance Centre (VISAVET-UCM).

Conflicts of Interest: The authors declare no conflicts of interest.

Appendix A

Appendix A.1

Table A1. Antimicrobial resistance genes and mechanism of the 13 Acinetobacter isolates.

ID of isolate	Genus and Specie	Antimicrobial resistance genes
1125	*A. pittii*	*amvA; nreB; tet(X3); sul2, sul1, merE, merD, merA, merT, merR; aph(6)-Id; aph(3″)-Ib; aac(3)-Iid; aac(6′)-Lb4; aph(3′)-Vla; abaF; qacEdelta1; arr-3; blaADC; blaOXA; blaCARB-16; msr (E); mph(E); drfA44*
1409	*A. baumannii*	*amvA; nreB; abaF; adeC; blaADC-166; blaOXA-106; ant(3″)-Iia*
1963	*A. baumannii*	*amvA; nreB; sul2; merT; merR; aph(6)-Id; aph(3″)-Ib; abaF; ant(3″)-Iia; aac(3)-Iie; aac(6′)-Ian; blaOXA-64; blaADC-26; parC_S84L; gyrA_S81L; tet(B); adeS_H189Y*
2080	*A. indicus*	*sul2; aph(6)-Id; aph(3″)-Ib; floR*
3321	*A. bereziniae*	*sul2; aph(6)-Id; aph(3″)-Ib; blaOXA; msr(E); mph(E); tet(B); floR; tet(39); ant(2″)-Ia*
3386	*A. radioresistens*	*blaOXA*
3556	*A. baumannii*	*amvA; nreB; abaF; ant(3″)-Iia; blaOXA-64; blaADC-26; adeS_H189Y*
3684	*Acinetobacter sp.* WCHAc010034	*blaOXA*
3904	*A. calcoaceticus*	*amvA; nreB; blaADC; blaOXA*
4255	*A. iwoffii*	*tet(X3); sul2; aph(6)-Id; aph(3″)-Ib; dfrA40*
4338	*A. baumannii*	*amvA; nreB; abaF; adeC; ant(3″)-Iia; parC_S84L; gyrA_S81L; blaADC-76; blaOXA-144*
4348	*A. baumannii*	*amvA; nreB; abaF; blaOXA; ant(3″)-Iia; blaADC-165*
4485	*A. bereziniae*	*blaOXA*

References

1. Quinn, P.J., Markey, B.K, Leonard, F.C, FitzPatrick, E.S, Fanning, S., Hartigan, P.J. *Veterinary Microbiology and Microbial Disease*. **2011.** Willy-Blackweell
2. López, S., López-Brea, M. *¿Qué debemos saber acerca de las infecciones por Acinetobacter baumanii?* **2024**. Enfermedades Infecciosas y Microbiología Clínica
3. Benga, L., Feßler, A.T., Benten, W.P.M, Engelhardt, E., Köhrer, K., Schwarz, S. y Sager, M. *Acinetobacter species in laboratory mices: species survey and antimicrobial resistance*. Laboratory Animals. **2019**. 53, 470-477. DOI: 10.1177/0023677218818598/journals.sagepub.com/home/lan
4. Nocera, F.P., Attili, A.R. y De Martino. L. *Acinetobacter baumanii: Its Clinical Significance in Human and Veterinary Medicine*. **2021**. Pathogens; 10, 127. DOI: https://doi.org/10.3390/pathogens10020127
5. Lee, C-R., Lee J.H.,Park, M., Park, K.S., Base, I.K, Kim, Y.B, Cha, C-J, Jeong B.C., Lee S.H. *Biology of Acinetobacter baumannii: Pathogens, Antibiotic Resistance Mechanisms, and Prospective Treatment Options*. **2017**. Frontiers in Cellular and Infection Microbiology. DOI: 10.3389/fcimb.2017.00055

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026 doi:10.20944/preprints202603.2022.v1

6. Doi, Y., Murray, G.L., Peleg, A.Y. *Acinetobacter baumanii: evolution of antimicrobial resistance-treatment options*; Semin Respir Crit Care Med. **2015**. 36, 85-98. DOI: 10.1055/s-0034-1398388
7. Van der Kolk, J.H., Endemiani, A., Graubner, C., Gerber, V. y Perreten, V. *Acinetobacter in veterinary medicine, with an emphasis on Acinetobacter baumanii*. **2018.** Journal of Global Antimicrobial Resistance. 16, 59-71. DOI: https://doi.org/10.1016/j.hgar.2018.08.011
8. Reina, R., León-Moya, C., Garnacho-Montero, J. *Tratamiento de infecciones graves por Acinetobacter baumannii*. **2022.** Medicina Intensiva. 46, 12. DOI: 10.1016/j.medin.2022.08.003
9. Naing, S.Y., Hordijk, J., Duim, B., Broens, E.M., van der Graaf-van Bloois, L., Rossen, J.W., Robben, J.H., Leendertse, M., Wagenaar, J.A. y Zomer, A.L. *Genomic Investigation of Two Acinetobacter baumanii Outbreaks in a Veterinary Intensive Care Unit in The Netherlands*. **2022**. Pathogens; 11, 123. DOI: https://doi.org/10.3390/pathogens11020123
10. Kuzi, S., Blum, S.E., Kahane, N., Adler, A., Hussein, O., Segev, G. y Aroch, I. *Multi-drug-resistance Acinetobacter calcoaceticus-Acinetobacter baumanii complex infection outbreak in dogs and cats in veterinary hospital*. **2016**. Journal of Small Animal Practice. 57, 617-625. DOI: 10.1111/jsap.12555
11. Al Bayssari, C., Dabboussi, F., Hamze, M., Rolain, J.M. *Emergence of carbapenemase-producing Pseudomonas aeruginosa and Acinetobacter baumannii in Livestock animals in Lebanon*. Journal Antimicrobial Chemother. **2015**, 70, 950–1. DOI: 10.1093/jac/dku469
12. Belmonte, O., Pailhoriès, H., Kempf, M., Gaultier, M.P., Lemarié, C., Ramont, C., et al. *High prevalence of closely-related Acinetobacter baumannii in pets according to a multicentre study in veterinary clinics, Reunion Island*. Veterinary Microbiology. **2014**.170, 446–50. DOI: 10.1016/j.vetmic.2014.01.042
13. European Comitee on Antimicrobial Susceptibility Testing. *Expected Resistant Phenotypes*. **2023**. Version 1.2.
14. World Health Organization. *WHO Bacterial Priority Pathogens List*. **2024**
15. Sebola, D.C., Oguttu, J.W., Kock, M.M. y Qekwana, D.N. *Antimicrobial resistance patterns of Acinetobacter baumanii and Klebsiella pneumoniae isolated from dogs presented at a veterinary academic hospital un South Africa*. **2023.** Veterinary World. 16, 1880-1888. DOI: www.doi.org/10.14202/vetworld.2023.1880-1888
16. World Health Organization. *One Health*. **2023**; https://www.who.int/news-room/fact-sheets/detail/one-health
17. Clinical & Laboratory Standards Institute; *Part B. CLSI vs FDA breakpoint*. **2023**; https://clsi.org/resources/breakpoint-implementation-toolkit/
18. Bolger A.M., Lohse M., Usadel B; *Trimmomatic: a flexible trimmer for Illumina sequence data. Bioinformatics*. 2014; 30:2114–2120. https://doi.org/10.1093/bioinformatics/btu170
19. Andrews S.; *Babraham bioinformatics - FastQC a quality control tool for high throughput sequence data*. 2010; https://www.bioinformatics.babraham.ac.uk/projects/fastqc/
20. Wood D.E., Lu J., Langmead B.; *Improved metagenomic analysis with Kraken 2*: Genome Biol. **2019**; Nov 28;20(1):257. DOI: 10.1186/s13059-019-1891-0.
21. Bankevich A., Nurk S., Antipov D., Gurevich A.A., Dvorkin M., Kulikov A.S., Lesin V.M., Nikolenko S.I., Pham S., Prjibelski A.D., Pyshkin A.V., Sirotkin A.V., Vyahhi N., Tesler G., Alekseyev M.A., Pevzner P.A.; *SPAdes: a new genome assembly algorithm and its applications to single-cell sequencing*. 2012; J Comput Biol 19:455–477. https://doi.org/10.1089/cmb.2012.0021
22. Gurevich A., Saveliev V., Vyahhi N., Tesler G.; *QUAST: quality assessment tool for genome assemblies*. 2013; Bioinformatics 29:1072–1075. https://doi.org/10.1093/bioinformatics/btt086
23. Parks D.H., Imelfort M., Skennerton C.T., Hugenholtz P., Tyson G.W.; *CheckM: assessing the quality of microbial genomes recovered from isolates, single cells, and metagenomes*. Genome Res. 2015; Jul;25(7):1043-55; DOI: 10.1101/gr.186072.114
24. Feldgarden M., Brover V., Gonzalez-Escalona N., Frye J.G., Haendiges J., Haft D.H., Hoffmann M., Pettengill J.B., Prasad A.B., Tillman G.E., Tyson G.H., Klimke W.; *AMRFinderPlus and the Reference Gene Catalog facilitate examination of the genomic links among antimicrobial resistance, stress response, and virulence*; Sci Rep. 2021 Jun 16;11(1):12728; DOI: 10.1038/s41598-021-91456-0
25. Hamouda, A., Findlay, J., Al Hassan, L., Amyes, S.G. *Epidemiology of Acinetobacter baumannii of animal origin*. **2011**. International Journal Antimicrobial Agents. 38, 314–8. DOI: 10.1016/j.ijantimicag.2011.06.007

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 25 March 2026
doi:10.20944/preprints202603.2022.v1

26. Plan Nacional Resistencia Antibióticos; *Categorización de los antibióticos;* https://www.resistenciaantibioticos.es/es/lineas-de-accion/vigilancia/antibioticos-criticos
27. Magiorakos, A.P., Srinivasan, A., Carey, R.B., Carmeli, Y., Falagas, M.E., Giske, C.G., Harbarth, S., Hindler, J.F., Kahlmeter, G., Olsson-Liljequist, B., Paterson, D.L., Rice, L.B., Stelling, J., Struelens, M.J., Vatopoulos, A., Weber, J.T., Monnet, D.L. *Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance*. **2012**. Clinical Microbiology and Infection. 3, 268-81. DOI: 10.1111/j.1469-0691.2011.03570.x
28. Migliaccio A., Bray J., Intoccia M., Stabile M., Scala G., Keith A., Brisse S., Zarrilli R.; *Phylogenomics of Acinetobacter species and analysis of antimicrobial resistance genes*; Volume 14 - **2023** | https://doi.org/10.3389/fmicb.2023.1264030
29. Cheng Y.Y, Liu Y., Chen Y., Huang F.M., Chen R.C., Xiao Y.H., Zhou K.; *Sporadic Dissemination of tet(X3) and tet(X6) Mediated by Highly Diverse Plasmidomes among Livestock-Associated Acinetobacter*: **2021**; Microbiol Spectr; Dec 22;9(3):e0114121. DOI: 10.1128/Spectrum.01141-21
30. Stoian I.A; *Multidrug-Resistant Acinetobacter baumannii: Resistance Mechanisms, Emerging Therapies, and Prevention—A Narrative Review;* **2025**; Antibiotics; 15(1), 2; https://doi.org/10.3390/antibiotics15010002
31. Álvarez-Buylla A., Culebras E., Picazo J.J.; *Identification of Acinetobacter species: Is Bruker biotyper MALDI-TOF mass spectrometry a good alternative to molecular techniques?*; **2012**; Infection, Genetics and Evolution 12 (345-349); https://www.sciencedirect.com/science/article/pii/S1567134812000032
32. Li X., Tang Y., Lu X.; *Insight into identification of Acinetobacter species by Matrix-Assisted Laser Desorption/Ionization Time of Flight Mass Spectrometry (MALDI-TOF MS) in the clinical laboratory*; **2018**; J. Am. Soc. Mass Spectrom; 29:1546Y1553; DOI: 10.1007/s13361-018-1911-4
33. Krishnaraj A., Gupta P., Bhatia M., Omar B.J.; *Role of Matrix-Assisted Laser Desorption Ionization-Time-of-Flight Mass Spectrometry for species identification of Acinetobacter strains*; **2023**; J. Lab. Physicians;15:336–343; DOI https://doi.org/10.1055/s-0042-1760401.

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.

© 2026 by the author(s). Distributed under a Creative Commons CC BY license.