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From soil to surface water: exploring *Klebsiella* 's clonal lineages and antibiotic resistance odyssey in environmental health

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Abstract

In the last decade, the presence of resistant bacteria and resistance genes in the environment has been a cause for increasing concern. However, understanding of its contribution to the spread of bacteria remains limited, as the scarcity of studies on how and under what circumstances the environment facilitates the development of resistance poses challenges in mitigating the emergence and spread of mobile resistance factors. Antimicrobial resistance in the environment is considered one of the biggest challenges and threats currently emerging. Thus, monitoring the presence of antibiotic-resistant species, in this particular case, *Klebsiella* spp., in the environment can be an added value for understanding the epidemiology of infections caused by *Klebsiella* spp.. Investigating soils and waters as potential reservoirs and transmission vehicles for these bacteria is imperative. Therefore, in this review, we aimed to describe the main genetic lineages present in environmental samples, as well as to describe the multidrug resistance strains associated with each environmental source. The studies analyzed in this review reported a high diversity of species and strains of *Klebsiella* spp. in the environment. *K. pneumoniae* was the most prevalent species, both in soil and water samples, and, as expected, often presented a multi-resistant profile. The presence of *K. pneumoniae* ST11, ST15, and ST147 suggests human and animal origin. Concerning surface waters, there was a great diversity of species and STs of *Klebsiella* spp. These studies are crucial for assessing the environmental contribution to the spread of pathogenic bacteria.

Keywords *Klebsiella* spp., *K. pneumoniae*, Antibiotic resistance, Soil, Surface water, Environment

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Background

Antibiotics are considered one of the greatest breakthroughs of the twentieth century. Since their discovery and subsequent commercialization, antibiotics have saved countless lives daily. Modern medicine depends heavily on its effectiveness in preventing and treating various infections, from common cases like urinary tract infections and pneumonia to severe, life-threatening conditions such as meningitis and sepsis [1]. However, almost simultaneously with their discovery, it was observed that bacteria could develop mechanisms to resist antibiotics. While antibiotics are highly beneficial for treating infections, promoting growth, and maintaining good health when used correctly, their widespread and indiscriminate use in both human and veterinary medicine over the years has led to a concerning issue – antibiotic resistance.

This overuse of antibiotics introduced a selective pressure that favored the development and evolution of resistance. Antibiotics have been used in situations with minimal or no therapeutic benefit, such as treating viral respiratory diseases or as growth promoters in livestock. During the COVID-19 pandemic over the past two years, antibiotics have been widely used to suppress infections or prevent secondary infections [2]. It has also been reported that up to 70% of COVID-19 patients have received antibiotics in outpatient or inpatient settings [3].

Currently, antibiotic-resistant bacterial infections are responsible for the deaths of 700,000 patients worldwide every year, including 230,000 due to multidrug-resistant (MDR) tuberculosis. If sustained efforts are not made, it is estimated that by 2050, the global death toll due to antibiotic-resistant disease-causing microorganisms could reach 10 million in low-, medium-, and high-income countries [4–6]. Although the ability of bacteria to develop antibiotic resistance is well-documented, the decrease in the development of new antibiotics and the increase in the spread of antibiotic-resistant bacteria have turned antibiotic resistance into one of the most significant threats to global public health. This issue is altering the landscape of modern medicine, bringing us closer to a pre-antibiotic era [7].

In recent decades, an increasing number of multidrug-resistant and even extremely drug-resistant (XDR) bacterial pathogens have emerged due to the overconsumption and reckless use of antibiotics, as well as the continued spread of mobile genetic resistance elements [8, 9]. Resistant infections are becoming increasingly challenging, if not impossible, to treat with current antibiotics. This trend results in infections causing greater morbidity and mortality, imposing substantial costs on society. Many common human pathogens, including *Enterococcus faecium*, *Escherichia coli*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and several *Enterobacter* species, have developed resistance mechanisms. Interestingly, many of these bacteria and their resistance mechanisms are found in the environment, including soil and water. The development of antibiotic resistance is not just a local public health concern; it is influenced by broader environmental factors and exacerbated by international travel and the global food trade [10].

Bacteria and their genetic material can move readily within and among humans, animals, and the environment. Microbial adaptations to antibiotic use and other selection pressures in one sector often affect others [11]. While the overuse and misuse of antibiotics remain significant drivers of resistance, other factors such as the movement of infected individuals across regions, inadequate infection control measures, and environmental contamination play pivotal roles in promoting the global spread of antimicrobial resistance (AMR) [12].

In recent years, the study of AMR has primarily focused on clinical pathogens, with the emergence of superbugs raising substantial public health concerns. However, due to the rise of zoonotic diseases and environmental contamination, the impact of AMR on animals, agricultural practices, wildlife, and the environment has garnered increased attention. This has led to collaboration across multiple sectors [13]. The result has been the formulation of the concept of "One Health," encouraging scientists and professionals from various disciplines to address the issue and its solutions in an interdisciplinary manner [14]. This concept is interdisciplinary and holistic, recognizing the interdependence of human and animal health within the ecosystems they inhabit [15, 16].

Consequently, AMR has been described as the "quintessential" issue of One Health, encompassing three main domains: humans, animals, and the environment. To mitigate the emergence and further spread of antibiotic-resistant organisms, the World Health Organization (WHO) has introduced policies focusing on antibiotic administration in healthcare settings and reducing antibiotic use in livestock production. However, to effectively manage antibiotic resistance, it is imperative to consider the broader environment. This involves gaining a better understanding of how human activities impact the development of antibiotic resistance, including pharmaceutical manufacturing waste, the release of household and agricultural waste into the environment, and the influence of poor sanitation and unsafe water supplies [10, 17].

Klebsiella spp. are widespread and can be found in environmental samples, including surface water, sewage, soil, and plants [18, 19]. Various strains of this genus have evolved to become significant clinical and public health threats worldwide. While *Klebsiella* spp. typically reside

in the nose, throat, skin, and intestinal tract of healthy individuals as opportunistic pathogens, they can also cause a range of infections, including pneumonia, soft tissue infections, surgical wound infections, urinary tract infections, bloodstream infections, and sepsis [20]. Consequently, it is crucial to investigate surface water and soils as potential reservoirs and transmission vehicles for these bacteria and their AMR determinants across ecosystems. This review aims to compile several recent studies on the prevalence of *Klebsiella* spp. in surface waters and soils, along with the genes found within them [21].

Antibiotic resistance in the environment

The consideration of the environment's role in the development and transmission of AMR is relatively recent, and actions in the environmental sector currently have the least implementation within the scope of public policies [17].

In recent years, the environment's role as an important source and route of resistance dissemination has been increasingly recognized. However, our understanding of its contribution remains limited. The scarcity of knowledge about how and under what circumstances the environment facilitates resistance development poses challenges in mitigating the emergence and spread of mobile resistance factors [22]. Understanding the environmental factors driving resistance can aid in constructing models depicting how resistance arises and spreads [23]. Although these models are initially descriptive due to many unknown parameters, they are crucial in identifying the most pressing knowledge gaps that must be addressed to develop effective mitigation strategies [24].

Hence, one of the six major emerging concerns recognized by the United Nations Environment Programme is antimicrobial resistance in the environment, alongside climate change, environmental degradation, and water stress. It is considered one of the global challenges humanity will face in this century [25]. However, a full understanding of the evolution, transmission, and persistence of antibiotic resistance genes remains elusive [13].

Some studies have reported the isolation of antimicrobial-resistant microorganisms from extreme and remote environments, largely untouched by modern antimicrobials and with minimal anthropic impact. This suggests that resistance may have emerged long before antibiotics were introduced into human activities [26].

Multiple environmental reservoirs contribute to the spread of AMR, including soil, water, medical facilities, industrial sites, agricultural areas, and various polluted ecological niches. The reckless use of antibiotics in both animals and humans, coupled with environmental contamination and inadequate infection control policies, are among the drivers of local and global AMR distribution [15].

Emerging concerns suggest that human impacts are altering environmental resistance gene reservoirs, known as "the resistome". This collection includes all resistance genes in a sample, regardless of the sample's origin. This increases the risk of resistance gene recruitment into clinically relevant pathogens. For instance, wastewater treatment, drug manufacturing, and agricultural effluents release significant amounts of antibiotic residues and resistant bacteria, which can subsequently enter the digestive tracts of humans or animals that use antibiotics [27].

Exposure of environmental bacteria to antibiotics, as well as to many resistant bacteria, can expedite the evolution of resistance and enhance the abundance and distribution of resistance genes within the resistome, a critical factor in resistance development [10].

Over the last decade, several studies have investigated the distribution and propagation of ARGs in various environmental compartments, such as soil [28], surface water [29], drinking water [30], and polar environments [31]. The spread of ARGs and bacteria in the environment occurs through dispersal and selection mechanisms.

Humans and animals are interconnected through the environment, emphasizing the importance of considering antibiotic resistance within the "One Health" perspective, and promoting global collaboration and interdisciplinary communication [10]. It recognizes that AMR is an ecological issue shaped by multifaceted interactions involving microbial populations from these three sectors [26].

It is estimated that as much as 75% of recently emerged or resurfaced human infectious diseases are zoonotic, originating from animals. One Health extends beyond this, encompassing environmental health alongside human and animal health. It emphasizes that, given the ever-increasing human population growth, climate change, heightened pollution, and depletion of Earth's resources, health, and other disciplines must coexist to secure the future health and well-being of humans, animals, and the environment [11].

Soil as a reservoir for AMR

Soil, one of the largest and most diverse microbial habitats on Earth, arguably contains the richest and most diverse populations of microorganisms and DNA sequences among all environments affected by human activity [14, 32].

Soil bacteria harbor ARGs that can also be found in human clinical pathogens and in emerging pathogens yet to be discovered. The fecal matter application to soil, primarily through agricultural practices, is a fundamental

human activity. However, it raises concerns regarding AMR and exerts a significant impact on the soil environment [33].

For soil improvement, organic amendments of animal origin (manure) or human origin (biosolids) are widely utilized as valuable sources of nutrients for agricultural production and organic matter. Another practice of growing concern, which contributes to the increasing antibiotic resistance in soil, is irrigation with reclaimed wastewater, that is also used for irrigating grass in urban or peri-urban green spaces [34, 35]. Depending on the treatment process, reclaimed wastewater may contain ARB and pharmaceutical residues, including antibiotics [36].

The use of manure as fertilizer in agriculture not only results in the production of antibiotic-resistant bacteria and ARGs, as previously mentioned but also leads to antimicrobials presence. Large amounts of antibiotics are not fully metabolized in the animal body and are excreted into the environment. Furthermore, there is concern that the widespread use of fungicides in agriculture may eventually lead to resistance, consequently decreasing the effectiveness of fungicides used to treat human infections caused by fungi or yeast. Due to the chemical, biological, and physical complexity of manure and biosolids, it is challenging to pinpoint the specific agent or class of agents that interact with soil microorganisms to promote AMR [37].

The presence of antibiotic residues in water and soil can alter the ecological balance of existing microbial communities, leading to selective pressure in favor of resistant bacteria, which, in turn, can become predominant and spread to other ecological niches [26]. Some studies suggest that bacteriophages, and viruses, spread ARGs more rapidly in fertilized soil in the presence of antibiotics than in their absence [14]. More recent work claims that soils, along with their associated mineral and organic constituents, influence the bioavailability of tetracycline, as measured by a bioreporter linked to ARG expression [38]. Moreover, antibiotic residues absorbed on clay mineral surfaces activate the bioreporter, suggesting that they may exert selective pressure for AMR [14, 39].

Water as a reservoir for AMR

In aquatic environments, bacteria naturally harbor numerous resistance genes, and it has been well-established that initially, antibiotic-sensitive pathogens can acquire resistance genes from environmental bacteria. Conversely, the overuse of antibiotics in human and animal, as well as in livestock farming, is giving rise to severe environmental and public health concerns. This can be attributed to the increased concentration of antibiotic residues in wastewater, fostering the development of antibiotic resistance in bacteria. Numerous studies have demonstrated that wastewater serves as a reservoir of ARGs, persisting in the effluents of various wastewater treatment plants even after filtration and disinfection [26, 40].

Hence, the occurrence of antibiotic-resistant bacteria in water is becoming an increasingly pressing concern. Furthermore, the existence of antibiotic-resistant bacteria serves as an indicator of antibiotic contamination in the respective aquatic environment. In general, water quality and safety are paramount for social development and ecological sustainability [41]. It is crucial to ensure access to clean and safe water for all, as waterborne diseases like diarrhea, cholera, gastroenteritis, and numerous antibiotic-resistant bacterial infections are primarily transmitted through water [42]. In regions like the western coast of Gujarat, for instance, several cases of enteric diseases have been documented, with recurring occurrences. This phenomenon may be attributed to transposable resistance elements that expedite the acquisition of resistance to various antibiotics [43]. When bacteria encounter antibiotics in wastewater, they employ these mechanisms to combat the drugs, not only developing resistance to a single drug but also to several antibiotics, transforming into multidrug-resistant bacteria [4, 40].

The transmission of antibiotic resistance from naturally resistant bacterial communities to non-resistant ones is a crucial aspect impacting human, animal, and ecosystem health. Therefore, the genetic foundation of AMR and how resistance disseminates from the environmental setting to clinical environments have become subjects of significant interest [24, 44].

Often, potentially pathogenic bacteria released into waters can carry ARGs, which are inserted into mobile genetic elements such as plasmids, transposons, and integrons, capable of spreading among bacterial communities inhabiting water bodies [45, 46]. Through the run-off of sewage, wastewater, and hospital effluents, surface water contamination can occur, leading to dispersion in the environment and transmission to humans and animals through water contact or even via wildlife. In some instances, wastewater from municipal, hospital, and pharmaceutical industries is improperly discharged into surface water [47].

Water plays a vital role for both humans and animals, serving for consumption, irrigation, and recreational activities. These waters are extensively used in agriculture due to their availability and rich nutrient content. However, they also pose a potential source of antibiotic-resistant microorganisms and ARGs that can be transferred to agricultural products. Unfortunately, even the most advanced drinking water treatment methods cannot

eliminate all antibiotics and ARGs, allowing their entry into water distribution systems [48].

Wastewater treatment plants receive ARGs and ARBs from domestic and clinical sources; however, the removal of these and most antibiotics is not entirely effective. Consequently, despite reducing the bacterial abundance in effluents, wastewater treatment, even when following legal recommendations, still results in the ongoing release of ARBs and ARGs into the environment. Unfortunately, the risks associated with the release of substantial quantities of ARB and ARGs remain poorly understood [49]. The study of the incidence and potential control of antibiotic resistance in WWTPs has gained importance following the discovery of antibiotic resistance in wastewater effluents. Moreover, the presence of mobile genetic elements (MGEs) involved in horizontal gene transfer (HGT), particularly plasmids and phages, along with genetic recombination elements in the wastewater metagenome, underscores the potential for the propagation of ARGs within and between different environmental compartments [14, 50].

Through the analysis of several articles, it becomes evident that there is an urgent need to accelerate public health research efforts to advance water sustainability technologies. Furthermore, additional studies are required to explore effective treatment and disinfection methods that can eliminate ARBs in wastewater treatment plants, given that environmental and public health risks present significant challenges [40].

Klebsiella spp.

Klebsiella spp. are Gram-negative, non-sporulating, non-ciliated bacilliform bacteria with a thick cell wall, contributing to their high virulence in vivo and the mucoid appearance of their colonies in vitro. They belong to the Enterobacteriaceae family and are rod-shaped, facultative anaerobic cultures, possessing a polysaccharide capsule that plays a substantial role in pathogenesis and preventing phagocytosis [51, 52].

Several species and subspecies of *Klebsiella* spp. have been identified. Among them, *Klebsiella pneumoniae* is considered the most clinically significant in humans and animals, followed closely by *Klebsiella oxytoca*. Both are regarded as opportunistic pathogens with considerable relevance in community-acquired infections and hospital settings (nosocomial). These infections are particularly severe in immunosuppressed individuals, such as those hospitalized for transplants, in intensive care (ICU), or neonatal units (NICU) [18].

Klebsiella spp. is classified as an opportunistic pathogen, and it is present in surface water, plants, soil, wastewater, and various other environments, depending on the phylogroup [53, 54]. It possesses the ability to adapt to oxygenated and non-oxygenated environments. This adaptability, coupled with its drug resistance, poses a risk of resistance genes being transferred to other microorganisms, especially those conferring resistance to carbapenems. It also carries the potential to cause superinfections and exacerbate primary infections in immunosuppressed individuals [55].

In terms of transmission to susceptible individuals, there are several routes, including transmission through contaminated food or water. Additionally, transmission can occur through contact between animals and people, between people, or through airborne droplets. The latter two pathways are the most common means of infection [54].

Antibiotic resistance has rapidly spread within *K. pneumoniae* and other members of the genus since at least the early 1980s. Over the past two decades, the emergence and dissemination of genes encoding carbapenemases have been particularly significant. These genes are typically carried on plasmids and fall into five main groups: $bla_{\text{OXA-48,}}$ bla_{KPC}, bla_{VIM}, bla_{NDM}, and bla_{IMP} [56].

Klebsiella spp. is a common cause of nosocomial infections, including pneumonia, meningitis, liver abscess, urinary tract infection (UTI), wound infection, bacteremia, and sepsis. The presence of virulence genes that encode factors enabling *K. pneumoniae* to evade the mammalian immune system is associated with its pathogenicity. In recent decades, *K. pneumoniae* has also been reported as a cause of community-acquired infections, including liver abscesses, endophthalmitis, and meningitis in otherwise healthy individuals [52]. According to Shiri et al., this organism is responsible for approximately one-third of all Gram-negative infections [57].

However, the frequent use of antibiotics in hospitals has led to antibiotic-resistant strains of *K. pneumoniae*, limiting treatment options for infections caused by *Klebsiella* spp. Due to the incomplete understanding of antibiotic resistance mechanisms in bacteria, particularly *K. pneumoniae*, treatment is not only highly challenging but may inadvertently contribute to the intensification of antibiotic resistance [51].

As previously mentioned, *Klebsiella* spp. has acquired significant antibiotic resistance due to the widespread acquisition of genes encoding enzymes like ESBLs and carbapenemases. The increasing prevalence of carbapenemase-producing (KPC) strains of *K. pneumoniae*, carrying the $bla_{\text{KPC-3}}$ gene encoding carbapenemases, poses a major public health threat. Carbapenem-class antibiotics are often the last line of defense against persistent Gram-negative infections [58].

All the studies mentioned above suggest that *Klebsiella* spp. is a common commensal in the healthy human microbiota, potentially serving as a reservoir for

infections. The phenotypic and genotypic characterization of *Klebsiella* spp. isolates from healthy individuals may contribute to a better understanding of the significance of *Klebsiella* spp. as a reservoir of potentially hazardous characteristics for human health [18].

In several studies, *Klebsiella* spp. has demonstrated its ability to acquire mutations and genetic elements that confer antimicrobial resistance and/or virulence characteristics. This leads to the emergence of convergent clones known as multidrug-resistant and hypervirulent *Klebsiella* spp. (MDR-hv). These strains are simultaneously hypervirulent and resistant to multiple antibiotics and are evolving to produce phenotypically novel variants [20].

As previously mentioned in this review, *Klebsiella pneumoniae* is a facultative anaerobic Gram-negative bacillus belonging to the group of pathogens grouped under the acronym "ESKAPE" (which includes *Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter*). This acronym refers to their ability to escape the bactericidal effects of antibiotics [58].

K. pneumoniae is considered the most clinically relevant species in humans and animals; however, it is often MDR, defined as resistant to three or more classes of antibiotics [59]. Nevertheless, *K. pneumoniae* isolates can acquire different mechanisms conferring resistance to commonly used antibiotics. Of particular concern and, consequently, the most common mechanisms, are broad-spectrum β-lactamases (ESBLs) and acquired *amp*C enzymes (qAmpCs). These enzymes confer antibiotic resistance in the cephalosporin and third-generation carbapenem classes, respectively [1, 59].

These enzymes are of particular concern because resistance caused by them can lead to reduced effectiveness or even failure of antimicrobial therapy. Moreover, carbapenems are considered a last-resort group of antibiotics for treating infections caused by MDR *Enterobacteriaceae* [1].

The global antimicrobial resistance (AMR) crisis faced by hospitals is driven by ESKAPE pathogens, as previously mentioned. These pathogens are responsible for most infections in hospitalized patients that are difficult to treat with antimicrobial therapy. Interestingly, these pathogens, part of this group, are environmental bacteria, particularly soil bacteria, or commensals that have co-evolved with antimicrobial-producing organisms over millennia. They give rise to opportunistic infections in hospitalized or immunosuppressed patients but are not otherwise pathogenic, meaning they do not cause diseases in healthy patients. However, there is usually a gap of several years between the clinical use of a drug and the appearance of relevant mobile AMR genes in populations of human pathogens. The accumulation of AMR in these organisms is mainly due to horizontal gene transfer (HGT) facilitated by plasmids and mobile genetic elements. Among Gram-negative pathogens, particularly in *K. pneumoniae*, there are hundreds of known mobile AMR genes subject to HGT, and this species has been associated with the initial reports of many AMR genes before their widespread dissemination [60].

Regarding the spread of AMR genes in *Klebsiella*, the spread of carbapenemase-producing *Klebsiella* strains, such as those with genes similar to bla_{KPC}, $bla_{\text{NDM,}}$ and $bla_{\text{OXA-48}}$, has become a global threat, as it is one of the main causes of outbreaks in healthcare settings, with a consequent increase in morbidity and mortality associated with nosocomial infections [61].

In addition to carbapenemase genes, ESBL genes, such as $bla_{\text{CTX-M}}$, bla_{SHV}, and bla_{TEM}, are also prevalent in *Klebsiella* spp, as mentioned above. *The* $bla_{\text{CTX-M-15}}$ *gene*, for example, is a widespread ESBL gene that confers resistance to cephalosporins and has been reported in several studies. Furthermore, the coexistence of these genes with carbapenemase genes on the same plasmids is worrying, as it increases the multiresistance of *Klebsiella* strains, thus reducing the effectiveness of currently existing therapies [62, 63].

These superbugs are becoming increasingly common, and new strains resistant to nearly all clinically important antimicrobials are emerging. Unfortunately, the pharmaceutical industry is not producing enough new antibiotics to match the high emergence of bacterial infections resistant to widely used antibiotics. One of the organisms causing increasing public health problems is *K. pneumoniae* [64].

Hence, the significant increase in the incidence of multidrug-resistant (MDR) and extremely drug-resistant (XDR) pathogens within the Enterobacteriaceae group poses a major economic challenge. These pathogens are predominant natural residents of the human and animal microbiome. Despite its significant clinical importance, there remains a lack of comprehensive information about *K. pneumoniae* [64].

Klebsiella spp. in environmental samples

The studies analysed were performed in 27 countries, between 2011 and 2023. Water, particularly surface water, is one of the most significant carriers of bacterial spread [65]. Aquatic environments, including lakes, rivers, and seawater, act as pathways for the introduction of antibiotic-resistant bacteria and their associated ARGs into natural ecosystems. Moreover, aquatic ecosystems integrated into urban water cycles are primary sources of AMR and ARGs in human-associated environments.

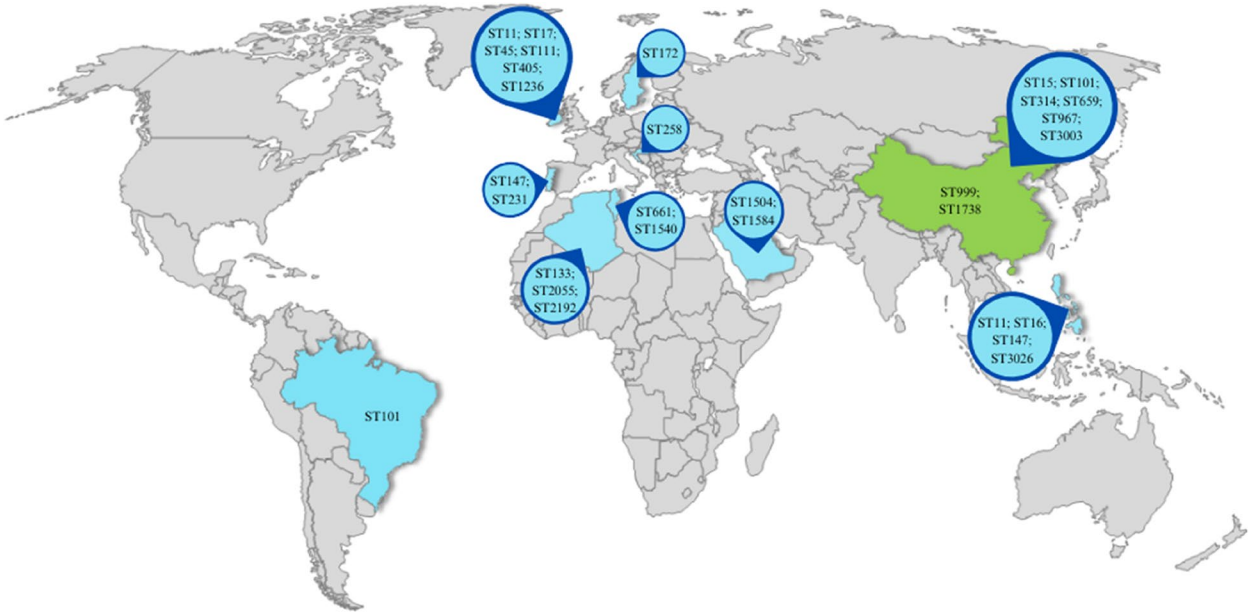

Fig. 1 Sequence types (STs) of *Klebsiella* spp. clones in different continents. Notes: Blue shading represents the water samples, while the green shading represents the soil samples

Figure 1 presents a general summary of the sequence types (STs) of *Klebsiella* spp. clones worldwide.

Some studies investigated the presence of *Klebsiella* spp. in soils (Table 1). A study carried out by Samanta et al., in soils from West Bengal, identified *K. pneumoniae* and *Klebsiella* spp.. In addition, some isolates exhibited resistance to amoxicillin + clavulanic acid, ceftazidime, cefalotin, cefpodoxime-proxetil, ceftriaxone, cefotaxime, and cefoxitin, encoded by the genes *ampC*, $bla_{\text{CTX-M}}$, bla_{TEM}, and bla_{SHV} [66]. Another study also carried out in soils in Tanzania analysed 97 soil samples, with 3 isolates of *K. pneumoniae* (3.1%) and resistance to antibiotics ceftriaxone, ampicillin, gentamicin, trimethoprim/sulfamethoxazole, nalidixic acid, tetracycline, imipenem, ciprofloxacin, and cefotaxime [67]. Chi et al. carried out a soil study in China, in which 23 samples were studied. From these samples, there was a prevalence of 9% of *K. pneumoniae* ($n = 2$), ascribed to STs, ST999 and ST1738. In addition, most isolates had a multidrug-resistance phenotype showing resistance to many antimicrobials such as amoxicillin + clavulanic acid, cefotaxime, gentamicin, tetracycline, ciprofloxacin, and trimethoprim/sulfamethoxazole, associated with ARR-3, *str*A, *str*B, *aph*(3′)-Ia, *aad*A16, *aac* [3]-IId, *qnr*S1, *oqx*A, *oqx*B, *fos*A, *mph*(A), *sul*2, *sul*1, *dfr*A27, *tet*(A), *flo*R, *aac*(6′)Ib-cr, $bla_{\text{CTX-M3}}$, $bla_{\text{SHV-1}}$, $bla_{\text{TEM-1B}}$, *dfr*A1, $bla_{\text{CTX-M-14}}$, and $bla_{\text{SHV-82}}$ [68]. In a study carried out in Peru, 83 soil samples were analysed, and among these samples, was reported resistance to amoxicillin + clavulanic acid, ampicillin, and cefoxitin [69]. Lastly, in a study carried out by Ebomah et al. 291 soil samples were analysed, with 38 isolates being identified as *K. pneumoniae* (13.1%). These isolates exhibited resistance to antibiotics imipenem, meropenem, ertapenem, and doripenem, and the prevalence of resistance genes $bla_{\text{NDM-1}}$, bla_{KPC}, and $bla_{\text{OXA-48-like}}$ was confirmed [70].

Regarding drinking water, in a study conducted by Samanta et al., in West Bengal, some resistance genes were identified including *aac*(6′)Ib-cr, $bla_{\text{CTX-M-3}}$, $bla_{\text{SHV-1}}$, and $bla_{\text{TEM-1B}}$ [66]. In another study conducted in Peru, some isolates revealed phenotypic resistance to ampicillin, ciprofloxacin, trimethoprim/sulfamethoxazole, and tetracycline [69].

At the West Bank, an investigation included influent and effluent greywater samples, among 49 samples from greywater influent, the isolates showed resistance to ampicillin, nitroxoline, and trimethoprim/sulfamethoxazole. On the other hand, from the 21 greywater effluent samples, was reported resistance to ampicillin, aztreonam, cefazolin, levofloxacin, nitroxoline, piperacillin, and trimethoprim/sulfamethoxazole. Notably, the prevalence of *Klebsiella* spp. was higher in greywater influents when compared to effluents [75].

Upon reviewing this article, we observed that the presence of *Klebsiella* spp. in river water was widely studied. This could be attributed to the increasing prevalence of bacteria in rivers, which, in turn, impacts bathing seasons due to the rising number of rivers with restrictions

Table 1 Distribution and diversity of *Klebsiella* spp. in soil and surface waters, and the genetic lineages of *Klebsiella* spp

Source	Location	Species	No. of isolates (%)	ST/CC	Resistance		Virulence genes	Reference
					Phenotype	Genotype		
Soil	West Bengal	*K. pneumoniae*	7/50(14%)		AUG, CAZ, CEF, CPD, CTR, CTX, FOX	bla_{CTX-M}, bla_{TEM}, *ampC*, bla_{SHV}		[66]
		Klebsiella spp.	7/50(14%)			bla_{CTX-M}, bla_{TEM}, *ampC*		[66]
	Tanzania	*K. pneumoniae*	3/97 (3,1%)		CTR, AMP, GEN, SXT, NAL, TET, IPM, CIP, CTX			[67]
	Northern New York	*K. oxytoca*	13/37 (35,0%)					[71]
		K. pneumoniae	4/37 (11,0%)					[71]
		K. variicola	2/37 (5,0%)					[71]
	China	*K. pneumoniae*	2/23 (9,0%)	ST999, ST1738	AUG, P/T, CTX, GEN, TET, CIP, SXT, FOS, F, FFC	ARR-3, *str*A, *str*B, *aph*(3')-Ia, *aad*A16, *aac*(3)-IId, *qnr*S1, *oqx*A, *oqx*B, *fos*A, *mph*(A), *sul*2, *sul*1, *dfr*A27, *tet*(A), *flo*R, *aac*(6')Ib-cr, $bla_{CTX-M-3}$, bla_{SHV-1}, bla_{TEM-1B}, *dfr*A1, $bla_{CTX-M-14}$, bla_{SHV-82}		[68]
	Peru	*Klebsiella* spp.	4/83 (4,0%)		AUG, AMP, FOX			[69]
	Iraq	*K. pneumoniae*	4/8 (50,0%)					[72]
	Ethiopia	*K. michiganensis*	2/16 (12,5%)					[73]
		K. oxytoca	1/16 (6,3%)					[73]
	Oman	*K. granulomatis*	1/16 (6,3%)					[74]
		K. oxytoca	1/16 (6,3%)					[74]
		Klebsiella spp.	1/16 (6,3%)					[74]
		K. pneumoniae	3/16 (18,8%)					[74]
	South Africa	*K. pneumoniae*	38/291 (13,1%)		IPM, MEM, ETP, DOR	bla_{NDM-1}, bla_{KPC}, $bla_{OXA-48-like}$		[70]
Drinking Water	West Bengal	*Klebsiella* spp.	1/1 (100%)			*aac*(6')Ib-cr, $bla_{CTX-M-3}$, bla_{SHV-1}, bla_{TEM-1B}		[66]
	Peru	*Klebsiella* spp.	4/27 (14,8%)		AMP, CIP, SXT, TET			[69]
Greywater Influent	West Bank	*Klebsiella* spp.	42/49 (71,2%)		AMP, NIT, SXT			[75]
Greywater Effluent	West Bank	*Klebsiella* spp.	4/21 (19,0%)		AMP, AT, CZ, LE, NIT, PIP, SXT			[75]
River Water	India	*K.pneumoniae*	25/33 (75,7%)		AMP, A/S, P/T, AT, CZ, FOX, CAZ, CTX, CTR, AK, C, CIP, PB, TR, S, LE, IPM, C, ETP	$bla_{TEM-206}$, bla_{SHV-38}, $bla_{CTX-M-55}$, bla_{SHV-11}, bla_{TEM-1}, $bla_{SHV-148}$, $bla_{TEM-116}$, bla_{SHV-1}, bla_{SHV-61}, $bla_{CTX-M-188}$, bla_{SHV-28}		[76]

Table 1 (continued)

Source	Location	Species	No. of isolates (%)	ST/CC	Resistance		Virulence genes	Reference
					Phenotype	Genotype		
		K. variicola	2/33 (6,1%)		AMP, CZ, P/T, AT, AK	$bla_{TEM-116}$		[76]
		K. oxytoca	1/33 (3,0%)		AMP, A/S, AT, CZ, FOX, CTX	$bla_{CTX-M-15}$		[76]
		K. quasipneumoniae	1/33 (3,0%)		AMP, A/S, CZ, CAZ, CTX, TR	$bla_{TEM-116}$		[76]
		Klebsiella spp.	4/33 (12,1%)		AMP, CL, TR, AT, CZ, CX, CTX, A/S, CAZ, PB, P/T, CTR	bla_{TEM-1}, $bla_{SHV-144}$, $bla_{CTX-M-15}$, bla_{SHV-27}		[76]
	Tunisia	*K.pneumoniae*	2/37 (5,4%)	ST661	NAL, SXT, S, C, TET	$bla_{CTX-M-5}$, bla_{SHV}		[77]
	Algeria	*K. pneumoniae*	3/20 (15%)	ST133, ST2192, ST2055	AC, AUG, ETM, FOX, TIC, FOS, CTR	bla_{OXA-48}, bla_{TEM-1}, bla_{SHV}		[78]
	Portugal	*K. pneumoniae*		ST231, ST147	PIP, P/T, TIC, TTC, AT, ETP, MEM, IPM, CAZ, CTX, CPM, CIP, SXT	bla_{KPC}		[79]
	Brazil	*K. pneumoniae*		ST101	AMP, AK, AT, CAZ, CEF, CIP, CTR, ETP, GEN, IPM, LE, MEM	bla_{TEM-1B}, bla_{OXA-1}, $bla_{SVH-182}$, $bla_{CTX-M-15}$, bla_{KPC-2}, *aac*(6)-Ib-cr, *gyr*A (S83Y), *gyr*A (D87A), *par*C (S80I), *aac*(3)-IIa, *aph*(3)-VIa, *cat*A1, *cat*A2, *cat*B3, *tet*(D), *dfr*A14, *fos*A		[80]
	Nepal	*Klebsiella* spp.	7/40 (17,5%)					[81]
		K. pneumoniae	5/40 (12,5%)			*tet*A, *sul*1		[81]
	South Africa	*K. pneumoniae*	7/40 (17,5%)		NIT, PB, AMP, AUG, AK, C, CIP, CS, CTX, CXM, DOX, NAL, NOR, SXT, TET	$bla_{OXA-48-like}$, bla_{SHV}, $bla_{CTX-M-2}$, bla_{TEM}, $bla_{CTX-M-1}$, bla_{ACC}, bla_{EBC}, bla_{FOX}, bla_{CIT}, *tet*A, *tet*B, *tet*M, *sul*1, *sul*2, *aad*A, *cat*2		[82]
		K. oxytoca	1/40 (2,5%)		NIT, PB, CL, AMP, AUG	bla_{ACC}, bla_{EBC}		[82]
	Iraq	*Klebsiella* spp.			CTX, AT, IPM, AC, AK, TR, GEN, CIP			[83]
		K. pneumoniae	35/101 (34,6%)		AMP, AUG, FOX, CTX, CEC, AK, IPM, MEM, CIP, NOR, FOX, CPR	bla_{NDM-1}		[84]

Table 1 (continued)

Source	Location	Species	No. of isolates (%)	ST/CC	Resistance		Virulence genes	Reference
					Phenotype	Genotype		
	Michigan	*K. pneumoniae*			CTX, AMP, AT, CZ, CPM, FOX, CTR, CXM, CIP, ETP, LE, CAZ, IPM, ETP, MEM, P/T, C, CIP, GEN	bla_{KPC}		[85]
	Tanzania	*K. pneumoniae*	8/97 (8,3%)		CTR, AMP, GEN, SXT, NAL, TET, IPM, CIP, CTX			[67]
	Sweden	*K. oxytoca*		ST172	CTX, CAZ, P/T, CIP, SXT	bla_{VIM-1}, bla_{OXA-10}, bla_{ACC-1}, *aac*(6')-Ib, *aad*A1, *str*A, *str*B, *qnr*S1, *sul*1, *dfr*A14		[86]
	Croatia	*K. pneumoniae carbapenemase (KPC)*		ST258	AMP, AUG, SXT, CIP, GEN, AK, all cephalosporins, all carbapenems	bla_{KPC-2}, bla_{SHV-1}, *aac*(3')-II, *aac*(6')-Ib and *aph*(3')-Ia		[87]
	China	*K. pneumoniae*	6/25 (24,0%)	ST967 ST15 ST101 ST3003 ST659 ST314	AUG, P/T, CTX, GEN, TET, CIP, SXT, FOS, F, FFC, CAZ	ARR-3, *str*A, *str*B, *aph*(3')-Ia, *aad*A16, *aac* (3)-IId, *qnr*S1, *oqx*A, *oqx*B, *qnr*B52, *fos*A, *mph*(A), *sul*1, *sul*2, *dfr*A27, *tet*(A), *flo*R, $bla_{CTX-M-3}$, bla_{SHV-28}, *qnr*B49, *dfr*A1, bla_{SHV-1}, bla_{SHV-11}		[68]
	Nigeria	*Klebsiella* spp.	4/26 (15,4%)		CXM, CTX, PEN, S, OXY, TET, NAL			[88]
	Ireland	*Klebsiella* spp.	1/28 (3,6%)	ST1236		$bla_{CTX-M-15}$, bla_{OXA-1}, bla_{SHV-1}	*mrk*A, *mrk*B, *mrk*C, *mrk*D, *mrk*F, *mek*H, *mrk*I, *mrk*J, *fyu*A, *irp*1, *irp*2, *ybt*A, *ybt*E, *ybt*P, *ybt*Q, *ybt*S, *ybt*T, *ybt*U, *ybt*X	[89]
	Philippines	*K. pneumoniae*	4/14 (28,6%)	ST16, ST147, ST11, ST3026	IPM, MEM, LE	bla_{NDM-7}, bla_{NDM-1}, bla_{KPC-1}, $bla_{CTX-M-15}$, $bla_{CTX-M-3}$		[90]
		K. oxytoca	2/14 (14,3%)		IPM, MEM, LE, GEN	bla_{NDM-7}, bla_{GES-20}		[90]
	Ghana	*Klebsiella* spp.	3/520 (0,6%)					[91]
	Poland	*K. pneumoniae*	33			bla_{GES}, bla_{VIM}, bla_{OXA-48}, bla_{TEM}, bla_{SHV}, bla_{OXA}, bla_{CTX}	*ybt*S, *mrk*D, *ent*B, K2, *kfu*, *allS*, *iut*A, *mag*A	[92]
Water	Iraq	*K. pneumoniae*	20/26 (77%)			bla_{SHV-1}	*mag*A, *rmp*A	[93]
		K. oxytoca	1/26 (3,8%)					[93]

Table 1 (continued)

Source	Location	Species	No. of isolates (%)	ST/CC	Resistance		Virulence genes	Reference
					Phenotype	Genotype		
	Saudi Arabia	*K. pneumoniae*	2/42 (4,8%)	ST1584, ST1504	AUG	*omp*K37, *omp*K36, *oqx*B, *oqx*A, *acr*R, *fos*A, bla_{SHV-1}, bla_{LEN16}		[94]
	Germany	*K. pneumoniae*	9			bla_{SHV-28}, $bla_{CTX-M-15}$, *tet*(D), *cat*B3, *aac* (3)-Iid, *str*A, *str*B, *fos*A, *ere*(A), *sul*1, *sul*2, *oqx*A, *oqx*B, *aac*(6')Ib-cr, *qnr*S1, *dfr*A5, *dfr*A14		[95]
Decanted Water	Tunisia	*K. pneumoniae*	1/37 (2,7%)		NAL, CIP, SXT, TET, S	$bla_{CTX-M-15}$, bla_{SHV}		[77]
Raw Water	Tunisia	*K. pneumoniae*	1/37 (2,7%)	ST1540	SXT, TET, S, C	$bla_{CTX-M-15}$, bla_{SHV}		[77]
Lake	Brazil	*K. pneumoniae*			AUG, CTX, CTR, CAZ, CPM, FOX, SXT, CIP, ETP, IMP, TIG	bla_{CTX-M}, bla_{KPC-2}, *oqx*A, *oqx*B		[96]
	Ireland	*Klebsiella* spp.	2/28 (7,1%)	ST111		$bla_{CTX-M-15}$, bla_{SHV-11}, bla_{TEM-1}, $bla_{TEM-1D-like}$, $bla_{OKP-B-3-like}$	*mrk*A, *mrk*B, *mrk*C, *mrk*D, *mrk*F, *mek*H, *mrk*I, *mrk*J, *fyu*A, *irp*1, *irp*2, *kfu*A, *kfu*B, *kfu*C, *ybt*A, *ybt*E, *ybt*P, *ybt*Q, *ybt*S, ybtT, *ybt*U, *ybt*X, *iut*A	[89]
Seawater	Ireland	*Klebsiella* spp.	4/28 (14,3%)	ST11, ST17, ST45, ST405		$bla_{CTX-M-15}$, bla_{TEM-1}, bla_{OXA-1}, bla_{SHV-11}, bla_{OXA-48}, bla_{SHV-1}, bla_{SHV-76}	*mrk*A, *mrk*B, *mrk*C, *mrk*D, *mrk*F, *mek*H, *mrk*I, *mrk*J, *fyu*A, *irp*1, *irp*2, *ybt*A, *ybt*E, *ybt*P, *ybt*Q, *ybt*S, *ybt*T, *ybt*U, *ybt*X, *kfu*A, *kfu*B, *kfu*C, *kvg*A, *kvg*S, *mce*A, *mce*B, *mce*C, *mce*D, *mce*E, *mce*G, *mce*H, *mce*I, *mce*J	[89]
Stream	Italy	*K. oxytoca*			AUG, AMP, CAZ, PIP, CTX, CPM, FOS	bla_{SHV-12}, $bla_{CTX-M-1}$		[97]
		K. pneumoniae			AUG, AMP, CAZ, CTX, CPM, PIP, CL, GEN	$bla_{CTX-M-1}$		[97]
	Ghana	*Klebsiella* spp.	33/520 (6,3%)					[91]
Dams	Ghana	*Klebsiella* spp.	28/520 (5,4%)					[91]
Irrigation Waters	Oman	*K. pneumoniae*	6/24 (25,0%)					[74]
		K. milletis	1/24 (4,2%)					[74]

Table 1 (continued)

Source	Location	Species	No. of isolates (%)	ST/CC	Resistance		Virulence genes	Reference
					Phenotype	Genotype		
		K. oxytoca	1/24 (4,2%)					[74]
	South Africa	*K. pneumoniae*	22/291 (7,6%)		IPM, MEM, ETP, DOR	$bla_{\text{NDM-1}}$, bla_{KPC}, $bla_{\text{OXA-48-like}}$		[70]
Surface Water	South Africa	*K. pneumoniae*	32/291 (10,9%)		IPM, MEM, ETP, DOR	$bla_{\text{NDM-1}}$, $bla_{\text{KPC,}}$ $bla_{\text{OXA-48-like}}$		[70]

Abbreviations: A/S Ampicillin/Sulbactam, *AUG* Amoxicillin + clavulanic acid, *AC* Amoxicillin, *AK* Amikacin, *AMP* Ampicillin, *AT* Aztreonam, *C* Chloramphenicol, *CAZ* Ceftazidime, *CEC* Cefaclor, *CEF* Cefalotin, *CIP* Ciprofloxacin, *CL* Colistin, *CPD* Cefpodoxime-proxetil, *CPM* Cefepime, *CPR* Cefpiroma, *CTR* Ceftriaxone, *CTX* Cefotaxime, *CXM* Cefuroxime, *CZ* Cefazolin, *DOR* Doripenem, *DOX* Doxycycline, *ETM* epithienamycins, *ETP* Ertapenem, *F* Nitrofurantoin, *FFC* Florfenicol, *FOS* Fosfomycin, *FOX* Cefoxitin, *GEN* Gentamicin, *IPM* Imipenem, *LE* Levofloxacin, *MEM* Meropenem, *NAL* Nalidixic acid, *NIT* Nitroxoline, *NOR* Norfloxacin, *OXY* Oxytetracycline, *P/T* Piperacillin/Tazobactam, *PB* Polymyxin B, *PEN* Penicillin G, *PIP* Piperacillin, *S* Streptomycin, *SXT* Trimethoprim/Sulfamethoxazole, *TET* Tetracycline, *TIC* Ticarcillin, *TIG tigecycline*, *TR* Trimethoprim, *TTC* Ticarcillin-clavulanic acid

imposed due to bacterial contamination. Moreover, many rivers are located in areas with cattle or irrigation, which could directly correlate with increased bacterial presence. The high prevalence of multidrug-resistant bacteria in rivers is concerning, a fact that is evident in the articles analysed in this review article.

In a study carried out in river water by Mondal et al. in India, there was a prevalence of 75.7% ($n=25$) of *K. pneumoniae*, 6.1% ($n=2$) *K. variicola*, 3% ($n=1$) *K. oxytoca*, 3% ($n=1$) *K. quasipneumoniae*, and 12.1% ($n=4$) *Klebsiella* spp.. Additionally, most isolates exhibited a multidrug-resistant phenotype showing resistance to many antimicrobials, such as ampicillin, ceftazidime, cefotaxime, ceftriaxone, imipenem, amikacin, ciprofloxacin, levofloxacin, trimethoprim, and carried the $bla_{\text{TEM-206}}$, $bla_{\text{SHV-38}}$, $bla_{\text{CTX-M-55}}$, $bla_{\text{SHV-11}}$, $bla_{\text{TEM-1}}$, $bla_{\text{SHV-148}}$, $bla_{\text{SHV-144}}$, $bla_{\text{TEM-116}}$, $bla_{\text{SHV-1}}$, $bla_{\text{SHV-61}}$, $bla_{\text{CTX-M-188}}$, $bla_{\text{CTX-M-15}}$, $bla_{\text{SHV-28}}$, and $bla_{\text{SHV-27}}$ genes [76]. Another study conducted in Tunisia reported a prevalence of 5.4% of *K. pneumoniae* mostly associated with ST661. Furthermore, these isolates exhibited resistance to nalidixic acid, trimethoprim/sulfamethoxazole, streptomycin, chloramphenicol, and tetracycline and carried the $bla_{\text{CTX-M-15}}$ and bla_{SHV} genes [77]. In Algeria, a study was carried out in river water ($n=20$), in which the prevalence of 15% of *K. pneumoniae* was detected and the strains belonged to ST133, ST2192, and ST2055. The isolates showed resistance to amoxicillin, amoxicillin + clavulanic acid, epithienamycins, cefoxitin, ticarcillin, fosfomycin, and ceftriaxone and carried the $bla_{\text{OXA-48}}$, $bla_{\text{TEM-1}}$, and bla_{SHV} genes [78]. A study conducted in the Lis River (Portugal), showed the presence of *K. pneumoniae* ST231 and ST147. These isolates also showed resistance to piperacillin, piperacillin/tazobactam, ticarcillin, ticarcillin + clavulanic acid, aztreonam, ertapenem, meropenem, imipenem, ceftazidime, cefotaxime, cefepime, ciprofloxacin, and trimethoprim/sulfamethoxazole. However, the only gene present in the isolates was bla_{KPC} [79]. In Brazil, a study carried out in a river passing through the Amazon, clonal strain ST101 was detected, and the strains showed resistance to ampicillin, amikacin, aztreonam, ceftazidime, cefalotin, ciprofloxacin, ceftriaxone, ertapenem, gentamicin, imipenem, levofloxacin, and meropenem and harbored the $bla_{\text{TEM-1B}}$, $bla_{\text{OXA-1}}$, $bla_{\text{SVH-182}}$, $bla_{\text{CTX-M-15}}$, $bla_{\text{KPC-2}}$, *aac* [6]-Ib-cr, *gyr*A (S83Y), *gyr*A (D87A), *par*C (S80I), *aac* [3]-IIa, *aph* [3]-VIa, *cat*A1, *cat*A2, *cat*B3, *tet*(D), *dfr*A14, *fos*A genes [80]. A recent study in Nepal reported a prevalence of *tet*A and *sul*1 genes [81]. In another study performed with river water from South Africa the isolates were multidrug-resistant and harbored the $bla_{\text{OXA-48-like}}$, bla_{SHV}, $bla_{\text{CTX-M-2}}$, bla_{TEM}, $bla_{\text{CTX-M-1}}$, bla_{ACC}, bla_{EBC}, bla_{FOX}, bla_{CIT}, *tet*A, *tet*B, *tet*M, *sul*1, *sul2*, *aad*A, *cat*2 genes [82]. In Iraq, a study carried out in river waters, reported resistance to cefotaxime, aztreonam, imipenem, amoxicillin, amikacin, trimethoprim, gentamicin, and ciprofloxacin [83]. In a study conducted in Michigan, were reported resistance to cefotaxime, ampicillin, aztreonam, cefazolin, cefepime, cefoxitin, ceftriaxone, cefuroxime, ciprofloxacin, ertapenem, levofloxacin, ceftazidime, imipenem, meropenem, piperacillin/tazobactam, chloramphenicol, gentamicin and carried the bla_{KPC} gene [85]. Kimera et al. conducted a study in river water from Tanzania, and all isolates were resistant to ceftriaxone, ampicillin, gentamicin, trimethoprim/sulfamethoxazole, nalidixic acid, tetracycline, imipenem, ciprofloxacin, and cefotaxime [67]. Another study conducted in Sweden reported the presence of *K. oxytoca* ST172 in river waters. In addition, the strains showed resistance to cefotaxime, ceftazidime, piperacillin/tazobactam, ciprofloxacin, and trimethoprim/sulfamethoxazole and carried the resistance genes $bla_{\text{VIM-1}}$, $bla_{\text{OXA-10}}$, $bla_{\text{ACC-1}}$, *aac*(6′)-Ib, *aad*A1, *str*A, *str*B, *qnr*S1, *sul*1, *dfr*A14 [86]. Jelic et al. conducted a study in Croatia where was verified the prevalence of *K. pneumoniae carbapenemase* (KPC). Clonal strain ST258 was identified, as well as resistance to ampicillin, amoxicillin + clavulanic acid, trimethoprim/sulfamethoxazole, ciprofloxacin, gentamicin, amikacin, all cephalosporins, and all carbapenems. Resistance genes included $bla_{\text{KPC-2}}$, $bla_{\text{SHV-1}}$, *aac*(3')-II, *aac*(6')-Ib, and *aph*(3')-Ia were also identified [87]. In a recent study in Iraq, the isolates showed resistance to ampicillin, amoxicillin + clavulanic acid, cefoxitin, cefotaxime, cefaclor, amikacin, imipenem, meropenem, ciprofloxacin, norfloxacin, cefpirome and carried the $bla_{\text{NDM-1}}$ gene [84]. In 2019, a study conducted in China reported the presence of *K. pneumoniae* ($n=6$; 24%), the isolates belonged mainly to ST967, ST15, ST101, ST3003, ST659, and ST314. In addition, the isolates showed a multidrug-resistant phenotype with resistance to amoxicillin + clavulanic acid, piperacillin/tazobactam, cefotaxime, gentamicin, tetracycline, ciprofloxacin, trimethoprim/sulfamethoxazole, fosfomycin, nitrofurantoin, florfenicol, ceftazidime associated with the ARR-3, *str*A, *str*B, *aph*(3′)-Ia, *aad*A16, *aac* (3)-IId, *qnr*S1, *oqx*A, *oqx*B, *qnr*B52, *fos*A, *mph*(A), *sul*1, *sul*2, *dfr*A27, *tet*(A), *flo*R, $bla_{\text{CTX-M-3}}$, $bla_{\text{SHV-28}}$, *qnr*B49, *dfr*A1, $bla_{\text{SHV-1}}$, and $bla_{\text{SHV-11}}$ genes [68]. A less recent study, conducted by Stanley et al. in Nigeria, reported the presence of *Klebsiella* spp. ($n=4$; 15.4%), along with resistance to the antibiotics cefuroxime, cefotaxime, penicillin G, streptomycin, oxytetracycline, tetracycline, and nalidixic acid [88]. Hoohan et al. reported the presence of *Klebsiella* spp. ($n=1$; 3.6%) in river waters from Ireland. These strains belonged to clonal strain ST1236. In addition, some resistance genes were identified including $bla_{\text{CTX-M-15}}$, $bla_{\text{OXA-1}}$, and $bla_{\text{SHV-1}}$. Furthermore,

isolates showed a wide diversity of genes encoding virulence factors, namely, *mrk*A, *mrk*B, *mrk*C, *mrk*D, *mrk*F, *mek*H, *mrk*I, *mrk*J, *fyu*A, *irp*1, *irp*2, *ybt*A, *ybt*E, *ybt*P, *ybt*Q, *ybt*S, *ybt*T, *ybt*U, and *ybt*X [89]. In the Philippines, a study conducted in river waters reported the prevalence of *K. pneumoniae* ($n=4$; 28.6%), which belonged to ST16, ST147, ST11, and ST3026. Moreover, resistances to imipenem, meropenem, levofloxacin, and gentamicin were detected and conferred by the $bla_{\text{NDM-7}}$, $bla_{\text{NDM-1}}$, $bla_{\text{KPC-1}}$, $bla_{\text{CTX-M-15}}$, $bla_{\text{CTX-M-3}}$, and *bla*GES-20 genes [90]. Lastly, a study in Poland, also in river water, identified *K. pneumoniae* ($n=33$). These isolates carried the resistance genes bla_{GES}, bla_{VIM}, $bla_{\text{OXA-48}}$, bla_{TEM}, bla_{SHV}, bla_{OXA}, and bla_{CTX}, and some virulence genes were identified including *ybt*S, *mrk*D, *ent*B, K2, *kfu*, *all*S, *iut*A, and *mag*A [92].

In this review article, we analysed three articles focusing on water samples. One of these studies collected water samples from Iraq and detected *K. pneumoniae* ($n=20$; 77%) and *K. oxytoca* ($n=1$; 3.8%). *K. pneumoniae* isolates harbored resistance gene $bla_{\text{SHV-1}}$ and virulence genes *mag*A and *rmp*A [93]. Another study in Saudi Arabia reported the prevalence of 4.8% of *K. pneumoniae* and the isolates were ascribed to ST1584 and ST1504. The isolates showed resistance to amoxicillin + clavulanic acid and carried the resistance genes *omp*K37, *omp*K36, *oqx*B, *oqx*A, *acr*R, *fos*A, $bla_{\text{SHV-1}}$, bla_{LEN16} [94]. The last study to analyse water samples was conducted in Germany, where the presence of *K. pneumoniae* was reported ($n=9$). These isolates carried the resistance genes $bla_{\text{SHV-28}}$, $bla_{\text{CTX-M-15}}$, *tet*(D), *cat*B3, *aac* (3)-Iid, *str*A, *str*B, *fos*A, *ere*(A), *sul*1, *sul*2, *oqx*A, *oqx*B, *aac*(6')Ib-cr, *qnr*S1, *dfr*A5, *dfr*A14 [98].

A study conducted in Tunísia reported a prevalence of 2.7% of *K. pneumoniae* among decanted and raw water. *K. pneumoniae* isolates from raw water were ascribed to ST1540. All isolates were resistant to trimethoprim/sulfamethoxazole, tetracycline, and streptomycin, however, isolates from decanted water were resistant to nalidixic acid, and ciprofloxacin, while isolates from raw water also displayed resistance to chloramphenicol. Finally, was reported the presence of $bla_{\text{CTX-M-15}}$ and bla_{SHV} genes [77].

Regarding the lakes, two articles were analysed. One of these studies was carried out in Brazil, in which the presence of *K. pneumoniae* was reported. The isolates showed resistance to amoxicillin + clavulanic acid, cefotaxime, ceftriaxone, ceftazidime, cefepime, cefoxitin, trimethoprim/sulfamethoxazole, ciprofloxacin, ertapenem, imipenem, and tigecycline, conferred by $bla_{\text{CTX-M}}$, $bla_{\text{KPC-2}}$, *oqx*A, and *oqx*B genes [96]. Another study carried out in Ireland reported the prevalence of *Klebsiella* spp. ($n=2$; 7.1%) with ST111 being the only sequence type detected. Finally, these isolates harbored the $bla_{\text{CTX-M-15}}$, $bla_{\text{SHV-11}}$, $bla_{\text{TEM-1}}$, $bla_{\text{TEM-1D-like}}$, and $bla_{\text{OKP-B-3-like}}$ genes, and a wide diversity of genes encoding virulence factors were also detected, namely, *mrk*A, *mek*H, *fyu*A, *irp*1, *kfu*A, *ybt*A, *iut*A, etc. [89].

In a study carried out in seawater in Ireland, there was a prevalence of *Klebsiella* spp. of 14.3% mostly associated with ST11, ST17, ST45, and ST405. Additionally, some resistance genes were identified, such as $bla_{\text{CTX-M-15}}$, $bla_{\text{TEM-1}}$, $bla_{\text{OXA-1}}$, $bla_{\text{SHV-11}}$. Regarding virulence, a high diversity of virulence genes were reported, such as *mrk*A, *mek*H, *fyu*A, *irp*1, *ybt*A, *kfu*A, *kvg*A, and *mce*A [89].

Regarding samples from streams, in a study carried out in Italy by Caltagirone et al., were reported the presence of *K. oxytoca* and *K. pneumoniae*. The isolates were resistant to amoxicillin + clavulanic acid, ampicillin, ceftazidime, piperacillin, cefotaxime, cefepime, and fosfomycin, and carried the resistance genes $bla_{\text{SHV-12}}$ and $bla_{\text{CTX-M-1}}$, in *K. oxytoca* isolates. On the other hand, *K. pneumoniae* isolates showed resistance to amoxicillin + clavulanic acid, ampicillin, ceftazidime, cefotaxime, cefepime, piperacillin, colistin, and gentamicin, and carried $bla_{\text{CTX-M-1}}$ gene [97]. Another study carried out in 2018 in Ghana reported a prevalence of *Klebsiella* spp. of 6.3%. In the same study, the authors reported the prevalence of 5.4% of *Klebsiella* spp. in dams [91].

In Oman, a study reported the presence of *K. pneumoniae* ($n=6$; 25%), *K. milletis* ($n=1$; 4.2%), and *K. oxytoca* ($n=1$; 4.2%) in irrigation waters [74]. Another study was also carried out in irrigation waters in South Africa, and there was a prevalence of *K. pneumoniae* of 7.6%. However, in the same study, the prevalence of *K. pneumoniae* was 10.9% in surface waters. This indicates a higher prevalence of *K. pneumoniae* in surface water than in irrigation water. Regarding resistance phenotype and genotype isolates from both sources showed resistance to imipenem, meropenem, ertapenem, and doripenem and carried $bla_{\text{NDM-1}}$, bla_{KPC}, and $bla_{\text{OXA-48-like}}$ genes [70].

As demonstrated in the aforementioned studies, STs were exclusively detected in soil samples, rivers, untreated water, lakes, and seawater. In the studies analysed within this article, the STs identified in soil samples were ST999 and ST1738, both of which were found in Chinese soils. Furthermore, in river waters, the most prevalent STs were ST147 and ST101. Conversely, similar to soils, water samples, untreated water, lakes, and seawater, the presence of STs was also reported in only one study. Therefore, due to the limited data available, we are unable to ascertain the predominant ST in these environments. Regarding resistance phenotype, in soil isolates the most prevalent resistance phenotypes were amoxicillin + clavulanic acid and cefotaxime and the greatest prevalent resistance genes were $bla_{\text{CTX-M}}$, bla_{TEM}, and

ampC. In rivers, the antimicrobials with the highest prevalence were cefotaxime, ampicillin, and imipenem. Additionally, the resistance genes $bla_{\text{CTX-M-15}}$, and bla_{SHV} were the most prevalent. The most frequently detected resistance genes in water were bla_{SHV}, *oqx*B, *oqx*A, and *fos*A. Finally, in streams isolates, the greatest prevalent antimicrobials were amoxicillin + clavulanic acid, ampicillin, ceftazidime, and cefotaxime, and the resistance gene that showed a higher prevalence was $bla_{\text{CTX-M-1}}$. A higher prevalence of *K. pneumoniae* was established in soil, river, water, and irrigation waters. On the other hand, in drinking water isolates, a higher prevalence of *Klebsiella* spp. was reported. Resistance among different sources analysed within this article is very high, particularly to cefotaxime and amoxicillin + clavulanic acid; the majority of studies referenced analyses the presence of these two antimicrobials in the samples under study. Therefore, the rise of antimicrobial resistance poses a major challenge for the medical world, affecting both human and veterinary medicine, especially in the treatment and management of infections caused by *Klebsiella* spp. and *K. pneumoniae*.

This review underscores the scarcity of studies investigating the prevalence of antibiotic resistance in *Klebsiella* spp. within the environment, particularly in the context of STs and virulence genes. Nonetheless, as previously mentioned, these studies hold paramount importance in assessing the environmental contribution to the dissemination of these bacteria and the potential exposure of humans and animals to them.

Conclusion

The environment is recognized as a reservoir of AMR and ARGs, even in the most restricted niches. Several studies have shown that it is important to reduce the use of fertilizers in agriculture, as they contribute to the increase in antibiotic-resistant bacteria and ARGs. Furthermore, as surface water is one of the main sources of water for human and animal consumption, its contamination can facilitate the spread of antibiotic-resistant strains of *Klebsiella* spp.. In addition, strains of *K. pneumoniae*, *K. oxytoca*, or broad-spectrum β-lactamase-producing species isolated from the environment are an important public health issue. However, data on the prevalence of antibiotic-resistant *Klebsiella* spp. in the environment remain limited, especially in the study of genetic lineages. These studies are crucial, since through them it is possible to estimate the environmental contribution to the dissemination of these bacteria to humans and animals.

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Authors' contributions
Conceptualization, S.A. and V.S.; validation, V.S. and P.P.; investigation, S.A. and M.Q; data curation, V.S. and A.M.; writing—original draft preparation, S.A.; writing—review and editing, S.A. and V.S.; supervision, V.S., G.I., and P.P. All authors have read and agreed to the published version of the manuscript.

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Data availability
All the data supporting our findings is contained within the manuscript.

Declarations

Ethics approval and consent to participate
Not applicable.

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