Climate-driven shifts in the growth and antimicrobial resistance of *Vibrio cholerae*, *Morganella morganii*, *Aspergillus niger* and *Trichoderma harzianum*

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Abstract. Antimicrobial resistance and climate change are two interrelated global health challenges that require urgent attention. Temperature, salinity and pH serve as indicators of climate change and can significantly influence microbial growth and antibiotic susceptibility. The aim of the present study was to explore the association between these parameters and antimicrobial resistance. For this purpose, the present study examined four different microorganisms: *Vibrio cholerae*, *Morganella morganii*, *Aspergillus niger* and *Trichoderma harzianum*. The results revealed a strong association between climate change conditions and antimicrobial resistance. For instance, *Vibrio cholerae* exhibited resistance to amikacin (10 μg), colistin (10 μg) and tetracycline (100 μg), while it exhibited increased sensitivity to piperacillin (100 μg) under specific environmental conditions, namely, a temperature of 27˚C, a pH of 10, and a salinity of 4%. Furthermore, *Aspergillus niger* developed resistance to ketoconazole (10 μg) and miconazole (50 μg) when grown in environments with a salinity level of 6%. On the whole, the present study demonstrates that microbial growth and antimicrobial resistant are affected by climate change- related factors, such as temperatures, salinity and environmental pH. These changes may contribute to the emergence of new strains harboring multidrug resistance genes.

Introduction

Environmental factors, such as temperature, salinity and acidity (pH) can affect the growth of microorganisms and their response to antibiotics. The spread of pathogens (vector-borne, waterborne, airborne and foodborne) and the occurrence of disease are sensitive to the effects of climate change. Fluctuations in temperature, rainfall, acidity, salinity and El Niño Southern Oscillation all affect the growth and spread of waterborne pathogens, such as *Vibrio* spp. in aquatic environments. Climate change is expected to increase the rate of antibiotic resistance in human pathogens by increasing the lateral gene transfer of mobile genetic resistance genes and increasing bacterial growth rates, promoting environmental persistence, carriage and transmission (1). The study by Mora *et al* (2) counted the number of microorganisms that have emerged and disappeared as a result of climate change. They discovered that 85% of the 357 infectious diseases controlled by humans have proliferated, whereas only 16% have decreased due to climate change (2). Climate change can cause disease spread for a variety of reasons, including the geographic expansion of vectors, such as mosquitoes, ticks, fleas, birds and other mammals as a result of rain, floods, or drought; these vectors transmit viruses, bacteria, fungi and parasites that have caused several diseases and epidemics, including dengue fever, plague, malaria, West Nile fever, cholera, trypanosomiasis, echinococcosis, and Nipah and Ebola viruses. The change in weather temperature has also placed individuals closer to pathogens and their habitats, such as increased water activities, which has increased the occurrence of waterborne illnesses such as cholera, primary amoebic meningoencephalitis and gastroenteritis, salmonellosis, shigellosis and others (2).

Another issue is Arctic ice melting due to global warming, which has resulted in the spread of Bacillus anthracis due to the reappearance of a previously frozen bacterial strain (3). In addition, the increasing rainfall has resulted in a decrease in water salinity, leading to the appearance of cases of cholera due to the increased quantity of *Vibrio cholerae* (*V. cholerae*) bacteria in the water (4). In some situations, climate change has negatively influenced particular types of diseases, causing their decline or elimination, such as, SARS, COVID-19, rotavirus and norovirus enteritis (5,6).

Generally, antimicrobial resistance and climate change are two of the top health crises; they are connected directly, and both affect human health by increasing the mortality rates. On the other hand, indirectly, climate change causes fluctuating temperatures, storms, forest fires and flooding. This has produced shifts in the ecosystem, increasing the transmission of vectors and infections and incorrect antibiotic usage, all of which lead to increased antimicrobial resistance (7).

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Numerous human pathogens, including *V. cholerae*, are naturally found in aquatic environments in rivers, coastal and estuarine habitats. *V. cholerae* is a key pathogen that causes cholera and has played a crucial role in human history. Its ability to survive in a variety of environments is largely due to the development of a set of adaptation strategies that enable these bacteria to survive under stresses such as nutrient deficiency, variations in salinity and temperature, and resistance to bacteriophage. Such a mechanism is the transformation to a viable but nonculturable state and has the capacity to grow as a biofilm on a variety of biotic (chitinous and gelatinous zooplankton and phytoplankton) and abiotic surfaces, allowing *V. cholerae* to disseminate to new watercourses through passive transfer or mechanical transfer (8).

Similar to *V. cholerae*, *Morganella morganii* (*M. morganii*) belongs to the *Gammaproteobacteria* class of Gram-negative rod bacteria, which are facultative anaerobic motile bacteria with flagella. *M. morganii* is widely distributed in the natural environment and as a normal flora in the intestinal tracts of humans and different animals. It is an opportunistic bacterium that clinically causes nosocomial infections in the urinary tract, hepatobiliary tract, skin and soft tissue, wounds and blood (9). Previously, little consideration was paid to these bacteria due to their rarity and low potential for causing nosocomial epidemics. However, *M. morganii* was then classified as a key pathogen due to its high mortality rate in some infections because of its virulence and elevated antibiotic resistance. This resistance is essentially acquired via extragenetic and mobile elements (10).

The impact of climate change on fungal infections is not yet fully known. Climate change is creating conditions that encourage the introduction of novel fungal infections and preparing fungi to adapt to previously hostile habitats. The present study selected two types of fungi: *Aspergillus niger* (*A. niger*) and *Trichoderma harzianum* (*T. harzianum*). *Aspergillus* is a genus in the order Eurotiales that has a global distribution and a diverse range of ecological habitats (11). Fungi are easily influenced by nutritional and physiological factors; therefore, even modest environmental changes can alter their morphological traits (12). In general, the dietary requirements for fungal growth are uncomplicated; nonetheless, various fungi require distinct physical, chemical, and nutritional environments. As a result, understanding the fungal habitat is contingent on knowing its temperature, hydrogen ion concentration (pH) and nutritional requirements. These fungi usually thrive in a laboratory at temperatures ranging from 0 to 40˚C. Additionally, the maximum metabolic activity, cellular growth, conidial generation and sporulation of *Aspergillus* spp. have been found in liquid conditions with an optimum pH range of 5-7 (13-15). Trichoderma, which belongs to the order Hypocreales, can be found in all climate zones. These fungi are typically found in soil and rotting wood (16). *T. harzianum* has extensive temperature, salinity and pH growth ranges, suggesting that it can adapt to changes in environmental conditions (17). The present study aimed to determine the effects of climate change on the growth rate and antibiotic resistance of microorganisms living in surface water.

Materials and methods

Sample collection and isolation of bacteria. Water was collected from various locations along the Tigris river in Baghdad, Iraq throughout the year. Water samples (250 ml) were collected in sterile containers and filtered through 0.22-μm membrane filters. For the isolation of *V. cholerae*, the membranes were then incubated in 100 ml alkaline peptone water (HiMedia Laboratories, LLC) overnight at 37˚C. A loop full of the overnight culture was streaked onto thiosulfate citrate bile sucrose (TCBS) agar (HiMedia Laboratories, LLC) and incubated at 37˚C overnight, as previously described (18). Yellow colonies were examined under a compound microscope (Olympus Corporation), and the comma-shaped colonies were then diagnosed after 6-7 h using a Vitek 2 compact system (Biomerieux). To isolate *M. morganii*, the membranes were incubated for 24 h at 37˚C in 100 ml nutrient broth (Oxoid; Thermo Fisher Scientific, Inc.). Subsequently, a loop full of overnight culture was streaked on MacConkey agar (HiMedia Laboratories, LLC), and the pale, non-lactose-fermented colonies were diagnosed using a Vitek 2 Compact system after 4-5 h, as previously descried (19).

Effects of different temperatures, pH value and salinities on bacterial growth. To examine the effects of different environmental conditions on bacterial growth in the laboratory, a 100-ml conical flask was used with a volume of 50 ml nutrient broth (Oxoid; Thermo Fisher Scientific, Inc.) and 1 ml inoculum at a concentration of $1.5x10^8$ CFU/ml, depending on the 0.5 McFarland standard. The flasks were incubated at different temperatures (17, 27 and 37˚C) for 24 h and aerated by shaking at 200 rpm to measure the effects of temperature on *V. cholerae* and *M. morganii* growth (20). The same conditions were used to examine the effects of various pH values (5, 6, 7, 8, 9 and 10) and different percentages of salinity (0, 2, 4, 6, 8, 10 and 14%).

Antibiotic susceptibility. The antibiotic susceptibility of *V. cholerae* and *M. morganii* was determined using the disk diffusion method on Mueller-Hinton agar (Oxoid; Thermo Fisher Scientific, Inc.), in accordance with the Clinical and Laboratory Standards Institute (CLSI) guidelines (21). The bacterial strains were examined under two sets of conditions as follows: i) For *V. cholerae*, the normal growth conditions were 37˚C, pH 8.6, and 2% salinity, whereas extreme conditions included 27˚C, pH 10 and 4% salinity; ii) for *M. morganii*, normal growth occurred at 37˚C, pH7, whereas extreme conditions were set at 27˚C, pH 10, 6% salinity.

The following antibiotics from (Bioanalyse) were used in the test: Tetracycline (30 μg), piperacillin/tazobactam (10 μg), ampicillin (25 μg), ampicillin (10 μg), colistin (10 μg), amoxicillin/clavulanic acid (30 μg), amikacin (30 μg); amikacin (10 μg), cefotaxime (30 μg), and piperacillin (100 μg).

Sample collection and isolation of fungi. Fungal isolates of *A. niger* and *T. harzianum* were obtained from the aquatic environment in Iraq (Tigris River). The isolation and cultivation were completed depending on established protocols (22). A total of 1 ml of each sample was aseptically inoculated

onto sterile 9-cm glass Petri dishes, and potato dextrose agar (PDA) medium (NEOGENLAB US) was then added with chloramphenicol (250 mg dissolved in 250 ml distilled water) to inhibit bacterial contamination. The plates were incubated at 25˚C for 48 h. The growing colonies were sub-cultured on PDA and incubated for 7 days at 28˚C. Microscopic (Optika Microscopes) examination and standard taxonomic keys were used to identify fungal species (23).

Of note, two methods were employed for the preparation of fungal inocula. First, spore suspensions were generated by the addition of 10 ml sterile distilled water to plates containing pure fungal colonies. A total of 5 ml of the resulting suspension were transferred into sterile glass bottles containing 95 ml sterile saline solution and were thoroughly mixed. Spore concentrations were determined using a dilution technique to achieve the desired inoculum density, as previously described (23,24). The final spore suspension densities were $1.7x10^3$ spores/ml for *A. niger* and $1.9x10^3$ spores/ml for *T. harzianum*. Alternatively, 7-mm-diameter mycelial discs were excised from actively growing fungal colonies and used in downstream applications.

Analysis of the effects of salinity, acidity and temperature. For the experiments, various concentrations of sodium chloride were used, including 2, 4, 6, 8, 10, 12 and 14%, which were added to the culture medium (PDA). Following sterilization in an autoclave at 121˚C and 1.5 bar pressure, the media were poured into sterile plastic petri dishes containing 1 ml chloramphenicol (HiMedia Laboratories, LLC) (250 ppm) and left to solidify. Subsequently, with a cork piercing a 7-mm size, each fungal isolate was removed and placed in a dish with a control, which was a Petri dish containing the antibiotic and culture media and fungus only. The Petri dishes were incubated at 28˚C for 2-7 days, after which the diameters of the developing colonies and the growth rates were measured. The previous steps used different pH values ranging from 5-10 to measure the effect of acidity on fungal growth. In the temperature experiment, three degrees were used: 17, 27 and 27˚C.

Antifungal resistance of A. niger and T. harzianum under different growth conditions. In total, five types of antifungals were used from (HiMedia Laboratories, LLC): Ketoconazole (KT), itraconazole (IT), amphotericin B (AP), nystatin (NS 50) and miconazole (MIC 50), and their effects on two fungal isolates (*A. niger* and *T. harzianum*) were investigated. PDA was prepared at different pH values and NaCl (HIMEDIA/India) concentrations (5 and 10%) and autoclaved at 121˚C and 1.5 psi. After cooling, the media were transferred to sterile plastic Petri dishes containing 1 ml spore suspension, as previously described (25), for each fungal isolate and 1 ml of the antibiotic, chloramphenicol (HiMedia Laboratories, LLC). They were allowed to firm up before the antifungal tablets were applied to each dish and incubated for 2-7 days at 28˚C.

Results

The growth and survival of microorganisms are influenced by their strains and a range of environmental factors, such as temperature and humidity, beneficial nutrients, pH, gas conditions and osmotic pressure (26). In the present study, to determine the effects of some of these parameters (TM, pH, and salinity) on different microorganisms, *V. cholerae* (Fig. 1) and *M. morganii* (Fig. 2) were selected as bacteria, and *A. niger* (Fig. 3) and *T. T. harzianum* (Fig. 4) were used as the fungi. All the microbes were isolated and diagnosed locally. As shown in Table I, it was found that *V. cholerae* could grow and survive at 4% salinity and *M. morganii* could grow in 6% NaCl media at 37˚C for 24 h of incubation; changing the acidity factor did not affect bacterial growth under the same incubation conditions. It was found that *V. cholerae* and *M. morganii* could survive temperatures ranging from 17 to 37˚C. When incubated under extreme growth conditions, *V. cholerae* thrived at 10 pH/4% NaCl/17˚C and 10 pH/4% NaCl/27˚C,

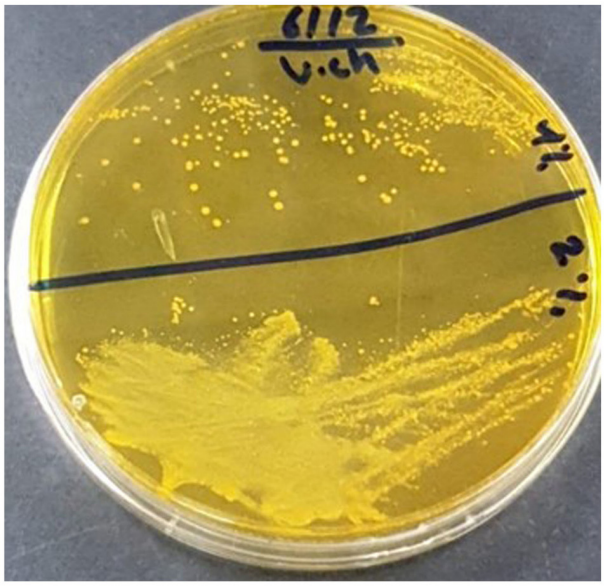

Figure 1. Colonies of *Vibrio cholerae* isolated from water samples growing on thiosulfate-citrate-bile salt (TCBS) agar.

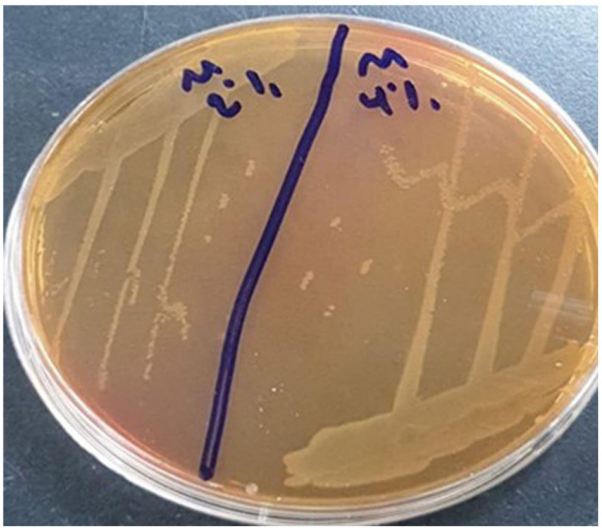

Figure 2. Colonies of *Morganella morganii* isolated from water samples growing on MacConkey agar.

Table I. Impact of environmental parameters on the growth of microorganisms.

	Growth of bacteria		Growth of fungi	
Variables Salinity (NaCl %)	*Vibrio cholerae*	*Morganella morganii*	Colony diameter(mm) of *Aspergillus niger*	Colony diameter (mm) of *Trichoderma harzianum*
0	+	+	77.5	82.5
2	+	+	75	55
4	+	+	60	25
6	-	+	50	12.5
8	-	-	45	-
10	-	-	30	-
12	-	-	-	-
14	-	-	-	-
Acidity (pH)				
5	+	+	75	78
6	+	+	55	78
7	+	+	60	78
8	+	+	60	79
9	+	+	65	79
10	+	+	65	79
Temperature,˚C				
17	+	+	-	-
27	+	+	75	80
37	+	+	-	-
pH/NaCl/TM				
10/4%/37˚C	+	+	-	-
10/6%/37˚C	-	+	-	-
10/4%/27˚C	+	+	-	-
10/6%/27˚C	-	+	30	-
10/10%/27˚C	-	-	-	-
10/4%/17˚C	+	+	-	-
10/6%/17˚C	-	+	-	-

The '+' symbol indicates that there was growth and the '-' symbol indicates that there was no growth.

whereas *M. morganii* survived at 10 pH/6% NaCl/17˚C and 10 pH/6% NaCl/27˚C.

The growth diameter of the fungi varied depending on the growth conditions. As demonstrated in Table I, it was found that *A. niger* could live at 10% salinity with a colony diameter of 30 mm, whereas *T. T. harzianum* could survive at 6% NaCl with a colony diameter of 12.5 mm. Both fungi flourished at various acidity levels, reaching a pH of 10. The effects of temperature on fungal growth were evident, and the fungi could not tolerate temperatures of 17 or 37˚C. When different variables were combined, only *A. niger* produced a 30-mm colony at pH 10, 6% NaCl, and 27˚C.

Antimicrobial resistance is a critical aspect of the recent century since the percentage of microorganisms that develop antibiotic resistance has increased rapidly. The present study used various types of antibacterial and antifungal agents under various growth conditions for four types of microorganisms. The findings presented in Table II demonstrated how environmental growth conditions can affect antibiotic resistance and antifungal resistance. *V. cholerae* developed resistance to amikacin (10 μg), colistin (10 μg) and tetracycline (100 μg) under extreme growth conditions, including pH 10, 4% NaCl and 27˚C, while it was intermediately resistant to amikacin (10 μg), and sensitive to colistin (10 μg) and tetracycline (100 μg) under normal growth conditions. Notably, *V. cholerae* became sensitive to piperacillin (100 μg) after growth under the same extreme conditions; however, its response to the other tested antibiotics remained unaltered. The resistance of *M. morganii* was largely unaffected by the altered growth conditions, apart from ampicillin (10 μg), to which it developed resistance under the same conditions.

As regards antifungal resistance, *A. niger* exhibited resistance to ketoconazole (10 μg) and miconazole (50 μg) under conditions of pH pH 10, 6% salinity and 27˚C; it also exhibited intermediate resistance to itraconazole (IT) 10 μg, amphotericin B (AP) 100 μg and nystatin (NS) 50 μg. Moreover, *T. T. harzianum* was resistant to amphotericin B (100 μg) and miconozole (50 μg) under the same conditions.

Figure 3. Colony of *Aspergillus niger* isolated from water samples growing on potato dextrose agar (PDA).

Figure 4. Colony of *Trichoderma harzianum* isolated from water samples growing on potato dextrose agar (PDA).

Discussion

Antibiotics and high or low temperatures are examples of environmental stressors that can alter the selection rules that exist in an ecosystem. These selection forces can have an impact on organism population evolution, as well as their ability to withstand environmental shocks (27,28). The present study revealed that bacterial strains exhibited greater resilience to diverse growth environments than fungal strains. The observed outcome may be attributed to the ability of bacteria to synthesize the sugar trehalose in response to different environmental factors, including low temperatures, oxidative stress, osmotic shock, acid stress and ethanol exposure (29).

Of note, environmental conditions can affect the external appearance and internal composition of bacteria. Nhu *et al* (30) found that variations in the acidity of the surrounding environment affected colony morphology, leading to the production of denser colonies with irregular edges, their results illustrate how bacterial morphology changes due to environmental stresses, which can affect their pathogenicity and identification in changing environmental contexts (30).

As regards the ability of *V. cholerae* to adapt to pH and temperature, the growth characteristics observed in the present study are consistent with those found in other published research. According to previous research, the optimal pH range for growth is between 6 and 9 (31-33), and the optimal temperature range is between 30 and 37˚C, although it can survive at 6˚C (34). However, the results of the present study make a noteworthy distinction by indicating salinity as a key determinant for the growth and environmental persistence of *V. cholerae*. It is important to note that at sodium chloride concentrations >4%, no growth was observed. Furthermore, while this observation regarding the effects of salinity contradicts the findings of Singleton *et al* (35) and Huq *et al* (31) regarding the growth of the cholera vibrio strain at higher concentrations of sodium chloride, it also suggests that strains may differ in their ability to withstand osmotic pressure; these changes in environmental factors can influence the conversion of environmental strains to pathogenic strains and trigger anew outbreak at any time (36). As regards *M. morganii*, the results were consistent with the findings of Frith *et al* (37) that this bacterium can tolerate various growth conditions of pH, salinity and temperature; it was isolated from all sites investigated, despite the different environmental circumstances.

The antimicrobial susceptibility of the microorganisms under investigation varied depending on the growth factors. Generally, at 27˚C, *V. cholerae* became resistant to amikacin and tetracycline. These results are in agreement with those in the study by Yuan *et al* (38), which reported that from 20 studies, 648 environmental *V. cholerae* isolates were investigated, where the weighted pool resistance rate for tetracycline and amikacin was 14 and 2%, respectively. Parvin *et al* (39) analyzed the data of tetracycline resistance from 2000 to 2018 and demonstrated that 100% of *V. cholerae* strains were susceptible to tetracycline between 2000 and 2004. Thereafter, a decline in susceptibility rate was observed, decreasing to 6% between 2012 and 2017, then increasing again to 76% in 2018 (39).

These medications (amikacin and tetracycline), which bind to ribosomes, have been demonstrated to either induce a cold-shock-like pattern of protein production or to interact with other stressors in a manner that is comparable to cold temperatures in *Escherichia coli* <22-27˚C. Given that one of the main effects of cold stress is translational block, the cellular machinery involved in response to these antibiotics may overlap with that involved in cold shock (40). When bacteria are repeatedly exposed to various growth environments, cross-tolerance may arise. This occurs when early exposure to a stressor leads to tolerance to a different type of stress. A previous study suggested that the mechanisms for tolerance may be shared among antibiotics that target similar processes rather than through generalized cell dormancy, creating multidrug-resistant bacteria (34). These shared tolerance

Table II. Influence of environmental growth conditions on the antimicrobial resistance profile of selected microorganisms.

A, Antibiotics (antibacterials)

Medication	Concentration	*Vibrio cholerae*		*Morganella morganii*	
		Normal conditions	Extreme conditions	Normal conditions	Extreme conditions
Amikacin (AK)	10 μg	I	R	I	I
Amikacin (AK)	30 μg	S	S	S	S
Ampicillin (AMP)	10 μg	R	R	I	R
Ampicillin (AMP)	25 μg	R	R	R	R
Amoxicillin/clavulanic acid (AMC)	30 μg	R	R	R	R
Cefotaxime (CTX)	30 μg	R	R	R	R
Colistin (CLM)	10 μg	S	R	R	R
Piperacillin (PRL)	100 μg	R	S	S	S
Piperacillin/tazobactam (PIT)	10 μg	R	R	R	R
Tetracycline	100 μg	S	R	R	R

B, Antibiotics (antifungals)

Medication	Concentration	*Aspergillus niger*		*Trichoderma harzianum*	
		Normal conditions	Extreme conditions	Normal conditions	Extreme conditions
Ketoconazole (KT)	10 μg	S	R	R	R
Itraconazole (IT)	10 μg	S	I	R	R
Amphotericin B (AP)	100 μg	S	I	I	R
Nystatin (NS)	50 μg	S	I	S	I
Miconozole (Mic)	50 μg	S	R	S	R

S, sensitive; R, resistant; I, intermediate.

mechanisms can also adapt to various types of stressors (such as temperature, pressure, or pH) (34).

After being subjected to a range of growth conditions, *V. cholerae* becomes resistant to colistin. Colistin is a last-resort antibiotic used to treat serious infections caused by Gram-negative bacteria (41). According to the study by Sharif *et al* (42), *V. cholerae* is typically susceptible to this antibiotic, with 59% of the 53 isolates being susceptible and 26% being resistant. As the resistance gene (mcr) is carried on a plasmid that may be transmitted from humans to animals, acquiring resistance to this antibiotic poses a new threat to human life by decreasing the effectiveness of eradicating harmful Gram-negative bacteria (41).

Drug resistance in Enterobacteriaceae pathogens (*V. cholerae* and *M. morganii*) is largely caused by the frequent transfer of extrachromosomal mobile genetic elements from nearby or distantly related bacterial species, even though chromosomal alterations can also play a role in antimicrobial resistance. The primary carriers of genetic characteristics expressing antimicrobial resistance function found in the isolates of enteric pathogens include plasmids, insertion sequences, superintegrons, integrating conjugative elements and transposable elements (43,44).

Antifungal resistance develops as a result of the changes that affect how the antifungal interacts with its target directly or indirectly. For example, mutations in genes encoding lanosterol demethylase (ketoconazole and miconazole) can cause changes in the binding site of the target that ultimately lead to resistance. Increased drug efflux activity for intracellular medications, such as azoles can increased target availability or modifications to the effective drug concentration can also lead to resistance (45,46). Polyene antifungals, such as amphotericin B and nystatin target ergosterol, an essential membrane component, rather than an essential cellular enzyme. This distinct mode of action could potentially account for the infrequent incidence of polyene resistance in fungi. However, changing the sterol makeup of the plasma membrane is the most well-known method of achieving polyene resistance (47). The findings of the present study may represent a basis for future investigations into the effects of climate factors on the phenotypic and genetic levels. Genetic mutations are widely known as key drivers of novel epidemics and diseases. Thence, it is recommended that following studies focus on the elucidation of the genetic impacts of these factors on microorganisms, which is essential for advancing the understanding of pathogenic and disease evolution.

In conclusion, the present study demonstrates that microbial growth and antimicrobial resistant are affected by changes in climate and climate-related factors, such as temperatures, salinity and environmental pH. These changes may contribute to the emergence of new strains harboring multidrug resistance genes.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

SAK was involved in the conception of the study, in the bacteriological work, in the analysis of the results, and in the writing of the manuscript. SHO was involved the fungal work, and in the writing of the fungal-related sections of the manuscript. Both authors have read and approved the final manuscript. SAK and SHO confirm authenticity of all the raw data.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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