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Review Article

Superbugs: antibiotic-resistant microorganisms, a public health problem

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Abstract
Superbugs are strains of bacteria resistant to several types of antibiotics. Their main characteristic is that they are capable of mutating their DNA throughout generations to become resistant to the most common drugs, which is why they represent a serious global health problem that has been contributed by the indiscriminate use of antibiotics, due to poor administration by the doctor, or by the self-medication to which the population frequently incurs, is added to this the pharmaceutical industry, the cattle ranch and the agriculture.
Objective: To carry out an updated review on the "superbugs", causes and mechanisms, and the importance of the emergence of a policy that regulates the indiscriminate use of antibiotics as well as the social and environmental factors that favor their emergence.
Material and Methods: A bibliographic search was carried out in different databases on the subject of microbial resistance and referring to "superbugs".
Results: Only the most relevant original articles were used with respect to the subject in question, whose date of publication was lower 10 years ago and from them, the most pertinent information was extracted regarding the subject that concerns us.
Conclusions: One must think about each and every one of the three causes that are at the origin of the problem (human, animal and industrial) with respect to the emergence of the "superbugs" since doing nothing will become one of the worst nightmares of Public Health in the years to come.

Keywords: superbusgs, multiresistance, antibiotic-resistance mechanisms

Introduction

The World Health Organization (WHO) describes "super bacteria" or "superbugs" as a group of Gram-negative bacteria that have the innate ability to resist treatments and have the ability to transmit genetic materials that allow other bacteria to become drug-resistant [1].

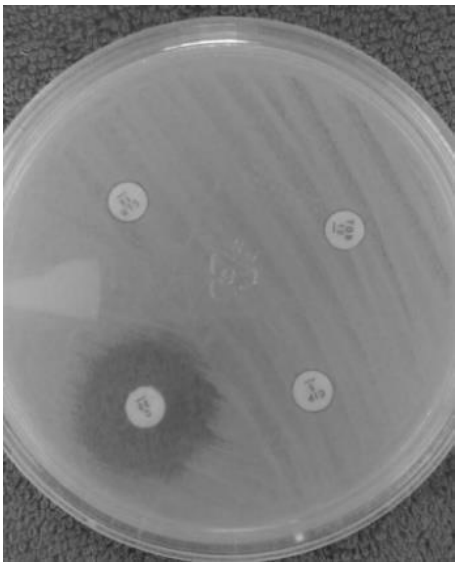

Figure 1. Multiresistance to antibiotics, Lab. Microbiology of Autonomous University of Guerrero, Mexico.

It is important to note that the term "super bacteria" is not synonymous with multidrug resistance because with multidrug resistance there exists a chance of developing a new drug, while when it comes to a "superbug" there is nothing that can be done to minimize their effects [2]. In Geneva, on the 27th of February 2017 WHO reported to the press the results of research conducted by experts from the Division of Infectious Diseases at the University of Tübingen, Germany, reporting that Antimicrobial Resistance is a serious problem throughout the world, and ranked the priority of the bacteria having an urgent need for development of new antibiotics as critical, high or medium [1] (Figure 1 showing multiresistance).

Superbugs, according to the WHO, are responsible for 700,000 deaths annually in the world representing a loss of between 2% and 3.5% of GDP. Should this trend continue it is expected 10 million deaths in the year 2050 [3]. In Latin America countries such as Bolivia, Cuba, Ecuador, El Salvador, Honduras, and the Dominican Republic are working in partnership with the United Nations Organization for Food and Agriculture (FAO) with the purpose of mitigating the emergence of multidrug-resistant bacteria and limit their spread in the sectors of food and agriculture. This organization sees the emergence of multidrug-resistant bacteria as one "more urgent [threats] facing humanity today" and constitutes a priority problem for public health [4].

In Mexico, in 2010, the Secretary of Health (SSA) reported that up to 60 percent of Mexicans showed resistance to major antibiotics in the market, so that in August of this same year by an agreement published in the Official Journal of the Federation (DOF) was enacted for the sale of antibiotic under medical supervision and with a prescription only [5].

Mexico ranks fifth among countries with a high risk of antimicrobial resistance. According to studies carried out, *E. Coli* showed the highest resistance to aminopenicillins, Pakistan ranks first with 93%, followed by India with 92%, China 88%, Kenya 86% and Mexico with 85% [6].

"Superbugs" originated due to a series of phenomena ranging from inadequate medical prescription, self-medication, and other circumstances such as the agricultural practices and the generation of "superbug" through the soil and water by using the waste streams as sewage, animal manure, and runoff from agricultural. The environment is also key in the resistance to antibiotics because the bacteria that are in the soil, rivers and sea water can develop resistance by coming into contact with other resistant bacteria and disinfecting agents which are released because of human activity, therefore, humans and livestock are exposed to increasingly resistant bacteria [6].

The objective of the present research is to perform an updated review on the "superbugs" causes and mechanisms, and the significance of the emergence of a policy regulating the indiscriminate use of antibiotics as well as social and environmental factors that favor the emergence of these.

Material and Methods

A bibliographic search was carried out in different databases on the subject of microbial resistance and relating to "superbugs", a policy of antimicrobial use, environmental policies, pharmaceutical industry, agro-industry, and hospital-acquired infections or associated to healthcare. The search was conducted in a framework of the past 10 years. Texts that met a higher level of scientific evidence were selected. A total of 100 articles were obtained, of which, according to the interest of this article, only 40 were selected, from which the information was obtained.

What are Superbugs?

Superbugs are strains of bacteria that are resistant to several types of antibiotics. The resistance may be intrinsic as with bacteria that carry genes that allow them

to survive exposure to antibiotics, or acquired through natural selection such as random changes or mutations that occur in genes of individual bacterial cells and the gene that carry antibiotic resistance is passed between bacteria. The result is the creation of bacteria that carries resistance genes to multiple antibiotics, these bacteria are known as superbugs or super bacteria [7].

Superbugs are organisms resistant to different classes of antimicrobials tested in the microbiological examinations, including the restricted use by the Commission of Control of Infection Hospital (CCIH) and the considered of controlled use, according to the WHO; these multidrug-resistant microorganisms are threatening a number of treatments, additionally they increase the time that people are sick, increase the morbidity and mortality, and the use of costly treatments to the institutions [8].

But...why and how does a superbug arise?

Mechanisms of bacterial resistance

Antibiotic resistance can be natural (intrinsic), unique to each family, genus or bacterial species, or acquired which is variable and typical of a strain of a bacterial species. The bacteria can acquire these mechanisms of resistance due to their genetic variability either by mutations or by horizontal transfer of genetic material between bacteria of related (or different) species. The genes can be encoded in the genetic material, chromosomal or extrachromosomal (plasmids). This resistance is the one that leads to the failure of antibiotics [9].

The mechanisms of resistance to antibiotics can be grouped into four categories:

- Enzyme inactivation: bacteria express enzymes that can create changes in the structure of the antibiotic causing it to lose its functionality. The main mechanism of inactivation is hydrolysis, as occurs with the β-lactamases, but non-hydrolytic modifications can also occur, such as acetylation, adenylation or phosphorylation inactivating the antibiotic [10].
- Changes in the permeability of the outer membrane: through alterations of bacterial membranes result in changes in the lipid bilayer by altering the permeability. Alterations in the input of antibiotics that are energy-dependent, or by the presence of efflux pumps that increase the expulsion of antibiotics [11].
- Overproduction or alteration of the target site of the antibiotic for example, the alterations of the PBPS (penicillin-binding proteins) or hyperproduction of the enzyme dihydrofolate reductase. The emergence of new metabolic pathways: alternative routes to the target site of action of the antibiotic. Although there have been many efforts to chemically modify the formulations of the antibiotics the reality is that the evolution of the dynamics of the bacterial genome and the acquisition of new genes through processes of genetic recombination has made this strategy ineffective and inefficient [10].
- Efflux pumps. Operate by taking the antibiotic in the periplasmic space and expelling it to the outside preventing the antibiotic from reaching its site of action. This is the mechanism frequently used for Gram-negative bacteria [11].

Horizontal transfer of genes

At a genetic level bacterium can generate antibiotic resistance as a result of chromosomal mutations and exchange of genetic material from other bacteria or phages (viruses that use bacteria for their development and reproduction) through mechanisms such as [12].

- Conjugation: Exchange of genetic material between two bacteria (donor and receptor) through a sexual strand or physical contact between the two mediated by plasmids, conjugative elements that have the property to be transferred from one cell to other thanks to close contact between the two cells through a pore of conjugation or sex pilus. The plasmids are mobile genetic elements, circular in shape, and they possess the ability to self-replicate independent of the bacterium. They possess gene cassettes coding for bacterial resistance [13]. They also produce deletions and gene mutations which optimize the bacterial processes [14].
- Transformation: The transfer or incorporation by a bacterium of free-extracellular DNA originating from the lysis of other bacteria [14].
- It is the transfer of genetic material, DNA, from a bacterium to another by a virus that infects bacteria (bacteriophage). This virus can integrate into the bacterial genome and when transferred to another cell can carry part of the genome of this bacterium, and so transfers genes including antimicrobial resistance genes [13].
- Transposition: Movement of a section of DNA (transposon) that may contain genes for resistance to different antibiotics and other gene cassettes working together for the expression of a particular promoter [12].

Means and methods that favor the emergence of superbugs

Sediments of surface and marine waters

Antibiotics that bind to sediment particles delay their biodegradation and explain their long-term persistence in the environment. Antimicrobial agents are retained in the soil because of their association with soil chemicals [15].

It is often assumed that hospitals are the most prominent source of introduction of antibiotics and bacteria resistance to municipal wastewater, but it has been reported that hospital effluents contribute less than 1% of the total amount present and although they are not the main source of resistant bacteria in the environment aquatic environment, they do contribute to the generation of these [15].

Within this perspective, it is not strange to think that hospitals and more specifically their liquid discharges exert a selective pressure on the biota present in the recipient bodies that receive them, contributing to the selection of microorganisms with multiple resistance patterns to antibiotics, and either by the presence of antibiotics in the discharges or by the transmission of resistance factors towards the bacteria found in surface waters [16].

Veterinary Medicine

The levels of concentration of veterinary antibiotics in environmental contexts such as soil, water, and manure, as well as their destination and transport in the environment, bring a comparison of the ecotoxicity data available in organisms for some of these drugs [16].

The biggest risk to the health of consumers surrounding the use of antibiotics in animals is not given by the waste, but by the development of resistance in bacteria of the same animals. These resistances can lead to therapeutic failures in veterinary treatments, and to the risk of transfer of resistant bacteria from animals to man, or of genes carrying information that encodes resistance of bacteria from animals to human bacteria [17].

The animal and human gastrointestinal tract has been considered as the place of choice for resistance transfers. Other niches, however, begin to be considered as of great importance. Thus, the intestine of wild animals (rodents), pets, and especially fish; considering commercial farms for their production, they represent places where the phenomenon would occur on a large scale [18].

Farming

The uncontrolled administration of antibiotics inevitably leads to residual concentrations in the excrement. Therefore, it is not surprising to find antibiotic residues in agricultural soils either as a metabolite or original compound in manure, fertilizer and later in agricultural fields [15].

Although the transfer of antibiotic-resistant microorganisms directly from animals to humans is known, there aren't virtually studies describing the transfer of resistance to antimicrobials to vegetables or animals reared in plots treated with sewage sludge with an intermediate passage through the soil. However, this connection can be made when analyzing that feeding from crops and fields treated with urban sludge or from animals contributes to the acquisition of these different profiles of resistances. The presence of antibiotic resistance genes in agricultural soils treated with sewage sludge may be abundant, although the transfer capacity of these resistances may be very limited. In the case of vegetables grown in this type of soil, the only resistance to antibiotics appears in the plants when the period of time indicated by the regulations is not respected, from the time the sludge is applied until harvesting occurs [19].

Classification

The WHO classified in three categories the bacteria that urgently require new treatment and that are at risk of running out of one; giving them the following priority designation: critical, high or medium (see table 1). The critical priority group includes multi-resistant bacteria, which are alarming present in hospitals, nursing homes and among patients who need to be treated with devices such as ventilators and intravenous catheters. Such bacteria include the following: *Acinetobacter*, *Pseudomonas* and several *Enterobactericie* such as *Kleibsiella*, *E. coli*, *Serratia*, and *Proteus*. They are bacteria that can cause serious and often lethal infecions, such as bloodstream infections and pneumonia [1].

These bacteria have acquired resistance to a high number of antibiotics, such as carbapenems and third-generation cephalosporins (the best antibiotics available to treat multi-resistant bacteria) [7].

The second and third levels of the list – the high and medium priority categories – contain other bacteria that exhibit in increased drug resistance and cause common diseases such as gonorrhea or salmonella food poisoning [1].

Another classification developed by the Center for Disease Control and Prevention (CDC) organizes these super bacteria according to the impact they cause on

Public Health in three levels: urgent, serious and worrisome. Microorganisms with an urgent threat level are *Clostridium difficile*, *Enterobacteriaceae* resistant to carbapenem, *Neisseria gonorrhoeae* drug-resistant; this group has the potential to spread in the population and is a serious threat to public health, so it requires measures to limit its growth [7].

Microorganisms with a serious threat level are *Acinetobacter* multi-drug resistant, *Campylobacter* drug-resistant, *Candida* resistant to fluconazole, Extended spectrum β-lactamases (ESBLs), vancomycin-resistant *Enterococcus* multi-drug resistant *Pseudomonas aeruginosa*, drug-resistant non-toxic *Salmonella*, drug-resistant *Salmonella typhi*, drug-resistant *Shigella*, methicillin-resistant *Staphylococcus aureus* (MRSA), drug-resistant *Streptococcus pneumoniae*, drug-resistant tuberculosis (MDR and XDR). These are significant threats [20]. Some bacteria though not currently labeled as urgent have the potential to become a nightmare if adequate public health surveillance and prevention are not carried out in a timely manner. These are the bacteria categorized as the worrisome level of threat such as vancomycin-resistant *Staphylococcus aureus* (VRSA), *Streptococcus group A* erythromycin-resistant, and *Streptococcus group B* clindamycin-resistant [21].

What are these bacteria?

Next, we will briefly explain the mechanism action leading these bacteria to be considered in the list of global priorities of bacteria resistant to antibiotics.

The mechanism of resistance to vancomycin in enterococci is established due to the existence of nine vancomycin-resistant phenotypes that contain the van A, B, C, D, E, G, L, M and van N genes. With van A phenotype being the most predominant worldwide. The mechanism of action of vancomycin acts by binding to the dipeptide D-alanyl-D-alanine terminal of the precursor of the peptidoglycan, thus inhibiting the synthesis of the cell wall. The VanA protein is a ligase that, instead of synthesizing the terminal dipeptide D-Ala-D-Ala, synthesizes D-Ala-D-lactate with much lower affinity for vancomycin.27

Priority 1: critical

***Acinetobacter baumannii* resistant to carbapenems**

Is responsible for infections, such as sepsis, meningitis and urinary tract infections, and therefore has acquired great importance in the control of infections by health personnel [22].

The mechanisms of resistance involve the enzymatic degradation of drugs, the modification or protection of the pharmacological receptor and decrease in the permeability of proton pumps of antibiotics that often work synergistically. Resistance to carbapenems in *A. baumannii* has been globally related to numerous enzymes, including OXA-type carbapenemase (OXA-51, OXA-23, OXA-24, and OXA-58) and Metallo-β-lactamase (IMP and NDM) [21]. Oxacillin is a β-lactam group that preferentially hydrolyzes oxacillin on benzylpenicillin. In addition, they can effectively inactivate aminopenicillins, methicillins and, to a lesser extent, some first-generation cephalosporins [23].

The loss of membrane permeability, due to changes in the primary structure, or loss of CarO (outer membrane protein associated with carbapenem), is classified into two groups, CarOa and CarOb, where CarOb has been shown to be the double of specific for imipenem than for CarOa [21].

These are currently the causes that best explain the resistance to carbapenemics of *A. baumannii* [21].

***Pseudomonas aeruginosa* resistant to carbapenems**

Pseudomonas aeruginosa is one of the main opportunistic pathogens that cause a large number of nosocomial infections, including sepsis, pneumonia, urinary tract infection, and soft tissue infection. This species is frequently isolated in immunocompromised patients who underwent organ transplantation, invasive procedure, immunosuppressive therapy or intensive care [24].

P. aeruginosa is one of the most important bacteria with documented resistance to multiple classes of antimicrobials, including β-lactams, carbapenems, aminoglycosides, fluoroquinolones, and polymyxins [25]. Due to it is intrinsic and acquired antimicrobial resistance, only a few classes of antibiotics are effective for the treatment of *P. aeruginosa* infections. Among the various mechanisms of antimicrobial resistance is the production of carbapenemase, one of the most important mechanisms by which *P. aeruginosa* acquires resistance to carbapenemics. Many carbapenemases have been identified in *P. aeruginosa* [26].

***Enterobacteriaceae*, resistant to carbapenem and 3rd generation cephalosporin**

Among the different carbapenem-resistant organisms, carbapenemase-producing *enterobacteria* (CPE) are posing a threat to global health due to its hasty dissemination, made possible through the horizontal transfer of genes. Currently, the drugs that are most used to treat *Enterobacteriaceae* (CRE) infections resistant to carbapenem only include tigecycline, aminoglycosides and several older antibiotics such as Fosfomycin and colistin [27].

MCR-1 (“Mobile Colistin Resistance”) is the first plasmid-

mediated colistin resistance gene that can be transferred horizontally through plasmids. This gene codes for a phosphorylethanolamine transferase, whose effect is the modification of the lipid A of the bacterial outer membrane thus preventing the interaction of the bacterium with the polymyxins. The relevance of this gene lies in its high horizontal dissemination capacity [26].

Table 1. WHO list of priority pathogens for the I+D of new antibiotics

Priority 1: CRITICA
Acinetobacter baumannii, resistant to carbapenems
Pseudomona aeruginosa, resistant to carbapenems
Enterobacteriace, resistant to carbapenems, producers of ESBL
Priority 2: HIGH
Enterococcus faecium, resistant to vancomycin
Staphylococcus aereus, reistant to methicillin, with intermediate sensivity and resistance to vancomycin
Helicobacter pylori, resistant to clarithromycin
Campylobacter spp., Resistant to fluorquinolones
Samonellae, resistant to fluorquinolones
Neisseria gonorrhoeae, reistent to cephalosporin, resisntant to fluorquinolones
Priority 3: MEDIUM
Streptococcus pneumoniae, without sesivity to penicillin
Haemophilus influenza, resistant to ampicillin

Source: Own elaboration from the WHO publishes de list of bacteria for wich new antibiotics are urgently needed.

Priority 2: HIGH

Vancomycin-resistant *Enterococcus faecium*

E. faecium is among the main causes of infections such as septicemia, endocarditis, urinary tract infections, wound infections, neonatal sepsis, and meningitis [27].

The mechanism of resistance to vancomycin in enterococci is established due to the existence of nine vancomycin-resistant phenotypes that contain the van A, B, C, D, E, G, L, M and van N genes. With van A phenotype being the most predominant worldwide. The mechanism of action of vancomycin acts by binding to the dipeptide D-alanyl-D-alanine terminal of the precursor of the peptidoglycan, thus inhibiting the synthesis of the cell wall. The VanA protein is a ligase that, instead of synthesizing the terminal dipeptide D-Ala-D-Ala, synthesizes D-Ala-D-lactate with much lower affinity for vancomycin [27].

***Neisseria gonorrhoeae* or gonococcus resistant to cephalosporins**

Gram-negative aerobic bacteria is the causative agent of gonorrhea. Three genetic mutations cause a decrease in susceptibility and chromosomal resistance to b-lactam antibiotics [28].

- The penA gene encodes changes in penicillin-binding proteins type 2 PBP2 (penicillin-binding protein 2). This alteration in its active site leads to a loss of the affinity of this for the antibiotic. Alterations in PBP-2 and in PBP-1 decrease the affinity for penicillin and the susceptibility of the microorganism to penicillin [28].
- The mtrR gene generates on expressed flow pumps that expel cytoplasm molecules from the b-lactams, especially ceftriaxone molecules, changing this gene can also stimulate the penB mutation [29].
- The penB gene alters the outer membrane porins (porB1b), preventing the cephalosporins from penetrating the bacterial cell [29].

Methicillin-Resistant *Staphylococci* (MRSA)

Currently, the bacterium has generated a genetic variation, the mecA gene. This gene allows the bacterium to modify the structure of its penicillin-binding protein (PBP2a), which prevents methicillin from adhering to the receptor, thus preventing the drug from acting to block the transpeptidase enzyme, preventing the bacterium the ability to form the bacterial wall. This mechanism gives Staphylococcus aureus resistance against semi-synthetic penicillins (Methicillin and Oxacillin) and first and second generation Cephalosporins [26].

Some strains of *S. aureus* have decreased their sensitivity to methicillin due to the increase in the production of β-lactamases, which include the modification of PBP. This group has been categorized as Golden Staphylococcus with Resistance limited to Oxacillin (BORSA for its acronym in English). This group has little response to treatment with semi-synthetic Penicillins and cephalosporins but does respond to Imipenem (Carbapenem) [25].

H. pylori resistant to clarithromycin

The resistance of this bacterium is related to a change in the structure of the Subunit (23S rRNA); since this subunit is the pharmacological target of clarithromycin. The structural change of 23S rRNA is due to a single nucleotide polymorphism (SNP) of the 23S rRNA gene [30].

The mechanism of resistance for mutations (adenine $\rightarrow$ guanine, adenine $\rightarrow$ cytokine) at positions 2142 (A2142G, A2142C) and 2143 (A2143G) are associated with the loss of binding of clarithromycin to ribosomes.31 At present, this is of great importance in the pheno-

typic determination of resistant strains by means of PCR" [31]

***Salmonella spp.* resistant to quinolones**

The first-line antibiotic treatment in the US for *salmonella* is usually ciprofloxacin, a third-generation cephalosporin or folic acid pathway inhibitors can also be used. Fluoroquinolones inhibit two bacterial enzymes DNA gyrase and topoisomerase IV, these play an important role in DNA replication. The ternary topoisomerase-quinolone-DNA complex leads to the generation of a double strand of DNA; quinolones block the progress of DNA replication in this enzyme complex, resulting in bacterial DNA damage and bacterial cell death [32].

Most of these mutations occur in the quinolone resistance determining the region (QRDR). Mutations in the QRDRs of these genes may confer reduced susceptibility or resistance to fluoroquinolones, depending on the number of mutations acquired. A single point mutation in the chemical conformation of this region confers resistance to nalidixic acid and decreases susceptibility to ciprofloxacin. Two or more mutations in the gene or other genes of the topoisomerase can confer complete resistance to ciprofloxacin [33].

Priority 3: MEDIUM

***Streptococcus pneumoniae*, not susceptible to penicillin**

The pneumococcal resistance to β-lactams has been attributed to the existing alterations of penicillin-binding proteins (PBP). The first isolated case of pneumococcus resistant to penicillin was reported in 1967 from a patient in Australia, later more cases of resistant *pneumococci* have been described around the world [34].

β-lactam antibiotics exert their biological effects by interacting with PBPs. Currently, the resistance of β-lactams in pneumococci is mediated by altered PBPs, specifically PBP1a, PBP2x, and PBP2b. This increase in resistance to antibiotics has come to present a health problem in places like Morocco [34].

***Haemophilus influenzae*, resistant to ampicillin**

The *H. influenzae* bacterium encodes four PBPs, the one responsible for generating resistance against ampicillin is the PBP3 mutation encoded by the FTSL gene, which in turn includes several mutations around the functional areas of the protein presenting four types of mutations. The main mutations that confer resistance are those that decrease the affinity in the union between the ampicillin molecules and the PBP, even generating resistance against cephalosporins; due to this, the prevalence of reported cases of *H. influenzae* resistant to ampicillin in different parts of Asia has increased [35].

***Shigella* spp. Resistant to fluoroquinolones**

As previously mentioned, the genetic mechanism of the pharmacological resistance of this bacterium is commonly attributed to mutations in the quinolone resistance determining region (QRDR), which in Ultimately, the interaction between the antimicrobial and its target decreases [33].

Resistance to fluoroquinolones leads us to increasingly reduced treatment options, placing those populations that are vulnerable to present a higher risk of complications caused by this bacterium and hindering the efficient management of outbreaks. These resistance factors have placed *Shigella* strains resistant to fluoroquinolones in the global list of priority pathogens that urgently need antimicrobial development [34].

Discussion

According to studies conducted by a specialist in Europe, infections with super bacteria resistant to multiple antibiotics kill about 33,000 people each year in that nation. It is estimated that about 70% of the bacteria that can cause an infection are already resistant to at least an antibiotic that is commonly used to treat them. This has led to the evolution of "superbugs", which can evade one or multiple medications. It is one of the biggest threats facing medicine today due to variations in the number of cases and types of antibiotic-resistant bacteria that cause infections in different countries, prevention and control measures must be adjusted to national situations [36].

In 2015 WHO launched the alert in this situation, immediately engaged in this project were thousands of scientists who defined a Global Action Plan on antimicrobial resistance. This was the official starting point to launch various strategies that managed to reverse a situation that claims millions of lives a year [37].

The unnecessary use of drugs such as antibiotics makes the bacteria develop more and more resistance to the point of becoming a kind of unbeatable "superbugs" or "super bacteria". These are particularly dangerous to patients admitted to the Intensive Care Unit, with chronic diseases, surgically intervened, since general people with diseases of the important base when acquiring a multi-resistant bacterial infection takes longer to heal because it is more difficult to treat [38].

Aware of the seriousness of the problem, both the World Health Organization and the countries most affected by this global problem have launched major awareness campaigns in recent years with the aim of reducing the abuse of antibiotics. However, despite numerous international and national efforts, the presence of resistant bacteria in Mexico City, where the presence of

Helicobacter pylori, causing gastric cancer, has been alerted, in 2005, a source of supply from Xochimilco was recorded by the Institute of Ecology. In addition, in 2014 *Vibrio cholerae* was identified in a well in the Sierra de Huejutla, Hidalgo, Mexico [39].

The bacteria *Vibrio cholerae* and *Helicobacter pylori* have the capacity to develop a state of resistance when presented in an aqueous medium as a survival mechanism or if they are in an adverse environment. Thus, they remain active (metabolism, respiration, infective capacity, genetic transcription, protein synthesis, and biomass production), but they are not able to replicate in a culture medium, which impacts the water quality tests and generates imprecision in the traditional modes of pathogen measurement, false negatives and an underestimation of their presence [39].

In 2010 the consumption of antibiotics in Mexico amounted to 70.5 million boxes per year, of which the authorities estimated that around 40 percent was due to self-diagnosis and self-prescription. That year, it was decided that this type of medicine would only be sold under medical prescription, but after 24 months of the implementation of that measure, consumption had been reduced much less than expected [40].

In the study Asymptomatic carriers of resistant methicillin Staphylococcus aureus (MRSA) in fishermen and horticulturists in Guerrero, Mexico detected from 57 fishermen and 50 horticulturists, only four MRSA They were detected due to resistance to oxacillin and being cefoxitin positive. From the results obtained from 57 fishermen and 50 horticulturists, only four MRSA strains were detected in fishermen; detection of these strains in the environment could be important. The presence of *S. aureus* is of epidemiological significance since it can be transmitted from people to people, food handlers that carry, since S. aureus, producers of enterotoxins, cause food poisoning [41].

As a strategy, and in conjunction with international organizations, Mexico published this June in the Official Gazette of the Federation the National Strategy of Action against Resistance to Antimicrobials, which intends to perform a work for the conscious and responsible use of antimicrobials for the health of humans, animals and crops in order to reduce the spread of resistance to antibiotics in different sectors. Strains detected in the hospital environment have a negative impact on the health of hospitalized patients; therefore, microbiological environmental monitoring and the strategies implemented help maintain a higher-quality hospital environment and potentially reduce the risk of patients developing healthcare-associated infections (HAIs) [42].

Finally, "the importance of environmental microbiological monitoring, in which sampling taken mostly in intensive care units, operating rooms and other hospital areas considered high risk from different surfaces, show that the microorganisms found are of risk to health, the microorganisms identified are mostly *Staphylococcus aureus*, *Acinetobacter baumannii*, *P aeruginosa*, *Klebsiella pneumoniae*, *Staphylococcus* coagulase, *Kytococcus spp* and *Cladosporium spp*, *Penicillium* and *Aspergillus spp*, recommend continuous microbiological monitoring to identify timely cleaning and disinfection to avoid cross infections and multi-resistant strains" [43].

Conclusion

The situation is clear when it comes to the fight against antimicrobial resistance: we must think about each and every one of the three causes that are at the origin of the problem (human, animal and industrial) if we do not want to fight. It has already been observed that getting the pharmaceutical sector to assume its responsibilities becomes a difficult task and that both the institutional prevention campaigns and the information focus are focused on the misuse of antibiotics by doctors and patients and in the abuse of antibiotics in intensive agriculture.

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Artificial intelligence. The authors declare that no artificial intelligence was used in any of the sections.

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