The mechanisms of antibiotic resistance in foodborne pathogens.

Maria Shuva*

Department of Food Science and Technology, Ho Technical University, Ghana

Introduction

Foodborne pathogens pose a significant threat to public health worldwide, causing a range of illnesses and even fatalities. The effective treatment of these infections has relied heavily on the use of antibiotics. However, the emergence and spread of antibiotic-resistant foodborne pathogens have become a growing concern in recent years. Understanding the mechanisms of antibiotic resistance in these pathogens is crucial for developing effective strategies to combat this global health issue. In this, we will explore the various mechanisms by which foodborne pathogens acquire resistance to antibiotics. One of the primary mechanisms through which foodborne pathogens acquire antibiotic resistance is Horizontal Gene Transfer (HGT). HGT allows the transfer of resistance genes between different bacteria, facilitating the rapid dissemination of resistance traits [1, 2].

This process can occur through several mechanisms, including conjugation (direct transfer of genetic material between bacterial cells), transformation (uptake of extracellular DNA), and transduction (transfer of genes via bacteriophages). HGT plays a significant role in the development and spread of antibiotic resistance in foodborne pathogens. Foodborne pathogens can also develop resistance through spontaneous mutations in their own genetic material. Mutations can alter the target site of an antibiotic, rendering it ineffective. Additionally, mutations can modify the metabolic pathways or efflux pumps that control the entry and efflux of antibiotics, making the pathogens resistant to specific drugs. Once a resistant mutant emerges, it can survive and multiply in the presence of antibiotics, leading to the selection of antibiotic-resistant strains within the population [3, 4].

Efflux pumps are another vital mechanism of antibiotic resistance in foodborne pathogens. These pumps actively transport antibiotics out of bacterial cells, reducing their intracellular concentration and rendering them less effective. Efflux pump systems are highly efficient and can confer resistance to multiple classes of antibiotics simultaneously. Overexpression or mutations in these pump systems allow foodborne pathogens to survive and persist in the presence of antibiotics, contributing to the development of resistance. Certain foodborne pathogens possess enzymes that can modify or degrade antibiotics, rendering them inactive. For example, beta-lactamases are enzymes that can hydrolyze beta-lactam antibiotics, such as penicillins and cephalosporins, preventing their action on bacterial cell walls [5, 6].

Enzymatic inactivation provides foodborne pathogens with an effective defence mechanism against antibiotics, allowing them to survive and cause infections. Foodborne pathogens can form biofilms, which are complex communities of bacteria embedded in a self-produced matrix. Biofilms provide an ideal environment for the exchange of genetic material and promote the survival of resistant strains. Within biofilms, bacteria are more tolerant to antibiotics due to factors such as limited penetration of drugs and reduced growth rates. This protective mechanism contributes to the development and persistence of antibiotic resistance in foodborne pathogens. The widespread and sometimes indiscriminate use of antibiotics in these settings creates a selective pressure, favouring the survival and proliferation of resistant strains. In addition to treating infections, antibiotics are often used for growth promotion and disease prevention in animals [7, 8].

This practice provides an environment where bacteria can acquire and maintain antibiotic resistance genes, which can subsequently be transmitted to human pathogens through various routes, including the food chain. Co-selection occurs when the use of one antibiotic selects for resistance to other antibiotics as well, further exacerbating the problem of multidrug resistance Antibiotic resistance in foodborne pathogens is a significant public health concern that requires urgent attention. Understanding the mechanisms by which these pathogens develop resistance is crucial for developing effective strategies to combat their spread. Horizontal gene transfer, mutation and selection, efflux pump systems, enzymatic inactivation, and biofilm formation all play essential roles in the acquisition and maintenance of antibiotic resistance in foodborne pathogens. Addressing antibiotic resistance in these pathogens requires a comprehensive approach that includes prudent use of antibiotics in agriculture and human medicine, improved surveillance, and the development of alternative strategies for infection prevention and control [9, 10].

References

1. Alam M, Hasan NA, Sadique A, et al. Seasonal cholera caused by Vibrio cholerae serogroups O1 and O139 in the coastal aquatic environment of Bangladesh. Appl Environ Microbiol. 2006;72(6):4096-104.
2. Alam M, Sultana M, Nair GB, et al. Toxigenic Vibrio cholerae in the aquatic environment of Mathbaria, Bangladesh. Appl Environ Microbiol. 2006;72(4):2849-55.

***Correspondence to**: Maria Shuva, Department of Food Science and Technology, Ho Technical University, Ghana, E-mail: Maria.s@htu.edu.gh

***Received:** 24-Oct -2023, Manuscript No. AAFMY-23-120323; **Editor assigned**: 28-Oct -2023, PreQC No. AAFMY-23- 120323(PQ); **Reviewed**: 10-Nov -2023, QC No AAFMY-23-120323; **Revised**: 15-Nov -2023, Manuscript No. AAFMY-23-120323(R); **Published**: 22-Nov -2023, DOI:10.35841/aafmy-7.6.171*

***Citation:** Shuva M. The mechanisms of antibiotic resistance in foodborne pathogens. J Food Microbiol. 2023;7(6):171*

3. Giao MS, Azevedo NF, Wilks SA, et al. Persistence of Helicobacter pylori in heterotrophic drinking-water biofilms. Appl. Environ. Microbiol. 2008;74(19):5898-904.

4. Ramamurthy T, Yamasaki S, Takeda Y, et al. Vibrio cholerae O139 Bengal: Odyssey of a fortuitous variant. Microbes and infection. 2003;5(4):329-44.

5. Bhanumathi R, Sabeena F, Isac SR, et al. Molecular characterization of Vibrio cholerae O139 Bengal isolated from water and the aquatic plant Eichhornia crassipes in the River Ganga, Varanasi, India. Appl Environ Microbiol. 2003;69(4):2389-94.

6. Mayer EA, Tillisch K, Bradesi S. Modulation of the brain–gut axis as a therapeutic approach in gastrointestinal disease. Alime Pharmacol Thera. 2006;24(6):919-33.

7. Hawkins KG, Casolaro C, Brown JA, et al. The microbiome and the gut-liver-brain axis for central nervous system clinical pharmacology: Challenges in specifying and integrating in vitro and in silico models. Clinical Pharmacology & Therapeutics. 2020;108(5):929-48.

8. Awouters F, Niemegeers CJ, Janssen PA. Pharmacology of antidiarrheal drugs. Annu Rev Pharmacol Toxico. 1983;23(1):279-301.

9. Macke L, Schulz C, Koletzko L, et al. Systematic review: The effects of proton pump inhibitors on the microbiome of the digestive tract—evidence from next-generation sequencing studies. Alimen Pharmacol Thera. 2020;51(5):505-26.

10. Del Pozo-Acebo L, López de las Hazas MC, Margollés A, et al. Eating microRNAs: Pharmacological opportunities for cross-kingdom regulation and implications in host gene and gut microbiota modulation. Briti J Pharmacol. 2021;178(11):2218-45.

Citation: *Shuva M. The mechanisms of antibiotic resistance in foodborne pathogens. J Food Microbiol. 2023;7(6):171*