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RESEARCH ARTICLE

The effect of bacteria on healthy and carcinogenic cells

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ABSTRACT

There are only a few types of bacteria that cause diseases. These types are called pathogens, which negatively affect cancerous tumors through the transformation and development of some types of bacterial infections into malignant tumors. Bacteria inhabit the body naturally and peacefully can sometimes cause diseases. Bacteria can cause disease by producing harmful substances (toxins or toxins), invading tissues, or both. Some bacteria can trigger inflammation that can affect the heart, nervous system, kidneys, or digestive tract. Bacteria (such as *Helicobacter pylori*) can increase the risk of developing cancer. Some types of bacteria may be used as biological weapons. These include those that cause anthrax, botulism, plague, and tularemia.

Keywords: Cancer, Antibacterial, Healthy cell, Cancerous cells, Chemical antibacterial compounds

INTRODUCTION

According to the somatic mutation theory, which is the dominant theory of carcinogenesis accepted in the scientific community, DNA mutations and carcinogenic epigenetic mutations destabilize these processes by disrupting their programmed regulation, upsetting the balance between proliferation and cell death. As a result, cells divide uncontrollably and develop in the body by natural selection. There are a few mutations responsible for cancer and most other mutations have nothing to do with carcinogenesis. Inherited gene mutations may predispose individuals to cancer,[1,2] as well as environmental factors such as carcinogens and radiation play a role in the mutations contributing to its development, and random errors in the transcription of the normal DNA strand may lead to oncogenic mutations. Cancer requires a series of multiple mutations in some types of genes before a normal cell can turn into a cancerous cell, as 15 "driver mutations" and 60 "rider mutations" have been found in colon cancers.[3-5] Mutations in genes regulating cell division, programmed death, and DNA strand repair may cause uncontrolled cellular proliferation leading to cancer. Cancer, by its basic definition,[6] is a disease that affects the regulation of the growth of living tissues. For a normal cell to turn into a cancerous cell, its genes regulating growth and differentiation must be changed. Genetic and epigenetic alterations can occur at many levels, from gain or loss of entire chromosomes, to mutations in a single nucleotide in the DNA strand, or to suppression or activation of microRNAs that control the expression of 100–500 genes.[7-9] There are two main genetic categories affected by these changes, the first is oncogenes, which may be normal genes expressed by the cell at inappropriately high levels, or altered genes with new properties, and in both cases, the expression of these genes induces the emergence of malignant phenotypes of cancer cells.[10-12] The second is the tumor suppressor genes, which are genes that inhibit cell division and reduce their viability and remove carcinogenic properties. These genes are inhibited by genetic alterations that induce cancer.[13-15]

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GENETIC AND NON-GENETIC CAUSES OF SPLITS

There is a broad taxonomic scheme for various genetic alterations that contribute to the generation of cancer cells, and mutations constitute a large proportion of them, and they are alterations in the nucleotide sequence that forms the DNA of the genome.[16-18] There are many epigenetic changes that affect the expression or lack of expression of genes. Aneuploidy is defined as the presence of several abnormal chromosomes in the nucleus, and it is one of the genetic alterations different from mutations, as it may involve the gain or loss of one or more chromosomes due to errors in the process of mitosis. Wide-effect mutations involve either a deletion or duplication of a portion of a chromosome. Genetic duplication occurs when a cell acquires copies (often 20 or more) of a small chromosomal region that usually contains one or more oncogenes with adjacent genetic material.[19-21] A chromosomal translocation occurs when two abnormally separate chromosomal regions fuse at a predominantly gender locus. An obvious example of a chromosomal translocation is the Philadelphia chromosome, a chromosomal translocation between chromosomes 9 and 22 that occurs in chronic granulocytic leukemia, and results in the fusion protein BCR-abl, an oncogenic tyrosine kinase. Mutations at the smaller level include point mutations, deletion, and insertion mutations that may occur in a gene promoter and affect gene expression, or occur in the gene-coding sequence and alter the function or stability of the protein product.[22-26] A single gene may be defective as a result of the insertion of genetic material from a DNA virus or retrovirus into the genetic material of the cell, and this may lead to the expression of viral oncogenes in the infected cell and the cells resulting from its division.[27-30]

SOME INFECTIONS TURN INTO CANCEROUS TUMORS DUE TO BACTERIA

It is impossible to determine the primary cause of most specific cancers. In a few cases, a single cause can be identified, such as the herpes virus HHV-8 that causes all Kaposi's sarcomas, but with the help of cancer epidemiology and information techniques, it has become possible to determine the likely cause in many cases. Lung cancer is one example of this, as this cancer has several causes,[31-33] including tobacco consumption and radon gas. The incidence of lung cancer in men who smoke is 14 times higher than that of non-smokers, with a 93% chance of smoking being a cause of lung cancer in a current smoker, and a 7% chance of radon causing it in non-smokers exposed to the gas.[34] These statistical correlations allowed researchers to infer certain substances or identify certain cancer-causing behaviors. Tobacco smoking causes an increase in exogenous DNA damage, and this damage carries the risk of lung cancer caused by smoking. Among the more than 5000 compounds in tobacco smoke, there are genotoxic agents that cause genetic damage, both of which occur in the highest concentrations. The most powerful mutagenic agents include: Acrolein, methanol,[35] acrylonitrile, butadiene 1-3, acetaldehyde, ethylene oxide, and isoprene. The use of molecular biology techniques has helped characterize genetic and epigenetic mutations and chromosomal aberrations within a tumor, and there has been rapid progress in predicting the prognosis of some cancers based on the spectrum[36] of mutations, as nearly half of tumors have a defect in the p53 gene. This mutation is associated with poor prognosis because these tumor cells are less likely to die or experience programmed cell death when damaged during treatment. Telomerase mutations remove further barriers to cell division, increasing the number of divisions in tumor cells, and other mutations allow the growth of new blood vessels that feed the tumor or spread metastases to other parts of the body. Once the cancer is formed, it will continue to develop and give rise to new subspecies. A 2012 report stated that examination[37-39] of nine samples from single kidney cancer from nine different regions revealed 40 mutations common to all of them, 59 to some, and 29 to just one region.[40,41]

It is difficult to trace the lineage of cells in which DNA changes accumulate, but two sets of evidence suggest that a normal stem cell may be the origin of cancer. First, there is a significant positive association (Spearman's correlation coefficient = 0.81: $P < 3.5 \times 10\text{-}8$) between the risk of developing cancer in the tissue and the number of stem cell divisions that occur in the same tissue. This association applies to 31 carcinomas and extends to 105 divisions. This association means that dividing a normal stem cell once increases the risk of cancer in tissue by approximately 1×, dividing 1000 times increasing the risk of cancer by 1000×, and dividing 100,000 times increasing the risk of cancer 100,000×. This strongly indicates that "normal" stem cell division is the main initiating factor for cancer and indicates that the origin of cancer is due to healthy stem cells.[42-44]

Second, statistics show that most human cancers are diagnosed in centenarians, and a possible explanation for this is that cancers occur because of the accumulation of cellular damage over time. A strand of DNA is the only cellular component that accumulates damage throughout its life and a stem cell is the only cell that can transfer DNA from an egg to other cells later in life, and since other stem cell-derived cells do not retain DNA from their inception until a possible cancer occurs, we conclude that most cancers arise from normal stem cells. More than half of somatic mutations have been identified in pre-proliferative (in the region of the defective field) tumors during development of apparently normal cells, and it was expected that many of the epigenetic changes present in tumors occur in the pre-progressive field-defective stage.[45] The defective field in the colon may arise by natural selection of a mutated or epigenetic cell from among the stem cells arrayed at the base of the intestinal crypts on the inner surface of the epithelium. A mutated or epigenetic stem cell may replace other nearby stem cells through natural selection and this could be a patch of abnormal tissue. These subclones are indicated on the figure by four small spots of different colors within the original large yellow macula.[46,47]

CAUSES OF THE DEVELOPMENT OF INFLAMMATION

When bacteria have a parasitic relationship with other organisms, they are classified as pathogenic agents of the body. Pathogenic bacteria are considered a major cause of human death and disease and also cause infection of the following diseases: Tetanus, typhoid fever, diphtheria, syphilis, cholera, poisoning, leprosy, and tuberculosis. It also causes proven medical diseases that may be discovered later, as is the case with *Helicobacter pylori* (stomach germs) and gastric ulcer disease. They infect farm animals such as Jones' disease, mastitis, salmonella, and anthrax. Each type of disease has its own characteristics that enable it to interact with its receptors in the human body. Some organisms such as streptococcus can cause skin infections, pneumonia, meningitis, and septicemia, which produce shock and dilate and swell the vessels, causing death.[48] However, these organisms are also part of the human body and are usually found on the skin or in the nose without causing any disease at all. There are other organisms that always cause diseases in humans, such as rickettsia, which is a parasite, as it resides, grows, and reproduces inside the cells of other living organisms. Only one type of rickettsia causes typhus, while some cause Rocky Mountain spotted fever. In addition, there is the *Chlamydia phylum*, a parasite that grows and reproduces inside cells, containing species that can cause pneumonia and urinary tract infection and may be involved in coronary heart disease. Finally, some species such as *Pseudomonas aeruginosa*, *Burkholderia*, and *Mycobacterium avium* are pathogens.[49-52]

Bacterial infections are treated with antibiotics, which are categorized as "bactericidal" if they kill the bacteria, or as "bacteriostatic" if they prevent their growth[53-55] There are many types of antibiotics and each class suppresses a different process by the disease virus from being present in the host (disease receptor). An example of how antibiotics produce selective toxins is chloramphenicol and puromycin, which inhibit the bacterial ribosome alone and not the structurally different nucleolar ribosomes. Antibiotics are used in the treatment

of human diseases and in intensive farming to promote animal growth, which would contribute to the rapid development of antibiotic resistance to the bacterial population.[56] Infection of diseases can be prevented through sterilization procedures such as disinfecting the skin before injecting it with medical needles and providing appropriate care for the catheter. Surgical tools and dental clinic tools must also be sterilized to prevent them from bacterial contamination.[57] Disinfectants are used as bleaches to kill bacteria or other germs on surfaces to prevent contamination as well as reduce the risk of infection.[58,59]

CONCLUSIONS

Finally, we mention Oncoviruses, which are viruses that contain oncogenes, and are classified as carcinogenic because they stimulate the growth of neoplastic tissues in the host's body, and this process is referred to as viral transduction.

REFERENCES

1. Witte W. International dissemination of antibiotic resistant strains of bacterial pathogens. Infect Genet Evol 2004;4:187-91.
2. Vardiman JW, Thiele J, Arber DA, Brunning RD, Borowitz MJ, Porwit A, *et al*. The 2008 revision of the World Health Organization (WHO) classification of myeloid neoplasms and acute leukemia: Rationale and important changes. Blood 2009;114:937-51.
3. Cătoi C, Baba AI. Comparative Oncology. Ch. 17. Bucharest: The Publishing House of the Romanian Academy; 2007. p. 10.
4. Aljamali NM. Inventing of macrocyclic formazan compounds and studying them against breast cancer for the first time globally. Ann Pharma Res 2021;9:525-33.
5. Aljamali NM. Creation of innovated macrocyclic Sulfazan-Formazan compounds and linear Sulfazan-Formazan for the first time globally with their assay as antifungal. Biomed J Sci Tech Res 2021;40:32266-72.
6. Aljamali NM, Jawad S. Preparation, spectral characterization, thermal study, and antifungal assay of (formazane -mefenamic acid)-derivatives. Egypt J Chem 2022;65:4266.
7. Aljamali NM. Synthesis of antifungal chemical compounds from fluconazole with (pharma-chemical) studying. Res J Pharm Biol Chem Sci 2017;8:564-73.
8. Andersson DI. The biological cost of mutational antibiotic resistance: Any practical conclusions? Curr Opin Microbiol 2006;9:461-5.
9. Larson E. Community factors in the development of antibiotic resistance. Annu Rev Public Health 2007;28:435-47.
10. Aljamali NM. Effect of Conditions and Catalysis on Products. 1st ed. Europe: Eliva Press SRL; 2021.
11. Aljamali NM, Kam I. Development of trimethoprim drug and innovation of sulfazane-trimethoprim derivatives as anticancer agents. Biomed Pharmacol J 2020;13:613-25.
12. Krm I, Abdulabas HK, Aljamali NM. Invention of (Gluta. Sulfazane-cefixime) compounds as inhibitors of cancerous tumors. J Cardiovasc Dis Res 2020;11:44-55.
13. Matheus ME, de Almeida Violante F, Garden SJ. Isatins inhibit cyclooxygenase-2 and inducible nitric oxide synthase in a mouse macrophage cell line. Eur J Pharmacol 2007;556:200-6.
14. Abdmajed MN, Aljamali NM. Preparation of benzothiazole-formazane reagents and studying of (spectral, thermal, scanning microscopy, biological evaluation). Int J Pharm Res 2021;13:4290-300.
15. Mad M, Aljamali NM, Nadheema AA. Preparation, spectral investigation, thermal analysis, biochemical studying of new (oxadiazole -five membered ring)-ligands. J Glob Pharm Technol 2018;10:20-9.
16. Abdullabass HK, Jawad AM, Aljamali NM. Synthesis of drugs derivatives as inhibitors of cancerous cells. Biochem Cell Arch 2020;20:5315-22.
17. Jawad AM, Aljamali NM. Innovation, preparation of cephalexin drug derivatives and studying of (toxicity and resistance of infection). Int J Psychosoc Rehabil 2020;24:3754-67.
18. Hmed H, Aljamali NM. Preparation, characterization, antibacterial study, toxicity study of new phenylene diamine-formazan derivatives. Indian J Forensic Med Toxicol 2021;15:3102-12.
19. Aljamali NM, Azez HM. Synthesis and characterization of some new formazan-cefixime and study of against breast cancer cells. Ann Roman Soc Cell Biol 2021;25:8562-78.
20. Aljamali NM, Mahdi AK. Synthesis, identification and anticancer studying of heterocyclic-mefenamic drug via thiosemicarbazide. Ann Roman Soc Cell Biol 2021;25:8521-37.
21. Jawad F, Aljamali NM. Preparation, investigation and study of biological applications of tyrosine derivatives against breast cancer cells. Neuroquantology 2021;19:117-25.
22. Aljamali NM, Alfatlawi IO. Synthesis of sulfur heterocyclic compounds and study of expected biological activity. Res J Pharm Tech 2015;8:1225-42.
23. Alfatlawi IO, Nuha SS, Zainab MJ, Aljamali NM. Synthesis of new organic compounds via three components reaction with studying of (identification,

thermal behavior, bioactivity on bacteria of teeth). J Glob Pharma Technol 2017;11:157-64.
24. Aljamali NM, Jawd SM, Zainab MJ, Alfatlawi IO. Inhibition activity of (Azo-acetyl acetone) on bacteria of mouth. Res J Pharm Technol 2017;10:1683-6.
25. Jawad AM, Aljamali NM, Jawd SM. Development and preparation of ciprofloxacin drug derivatives for treatment of microbial contamination in hospitals and environment. Indian J Forens Med Toxicol 2020;14:1115-22.
26. Rasool SR, Aljamali NM, Al-Zuhairi AJ. Guanine substituted heterocyclic derivatives as bioactive compounds. Biochem Cell Arch 2020;20:3651-5.
27. Ar D, Kilpatrick AF, Cula B, Herwig C, Limberg C, *et al*. Transformation of formazanate at nickel (II) centers to give a singly reduced nickel complex with azoiminate radical ligands and its reactivity toward dioxygen. Inorganic Chem 2021;60:13844-53.
28. Milocco F, de Vries F. Widening the window of spin-crossover temperatur in bis (formazanate). Inorganic Chem 2021;60:2045-55.
29. Ali HA, Aljamali NM. Chalcone-heterocyclic derivatives (synthesis, spectral identification, microbial evaluation). Int J Pharm Res 2021;13:4234-42.
30. Thomadsen B, Nath R, Bateman FB, Farr J, Glisson C, Islam MK, *et al*. Potential hazard due to induced radioactivity secondary to radiotherapy. Health Phys 2014;107:442-60.
31. Shapira T, Pereg D, Lishner M. How I treat acute and chronic leukemia in pregnancy. Blood Rev 2008;22:247-59.
32. Abl M, Ali A, Aljamali NM. Synthesis, spectral, bio assay, chromatographic-studying of new imidazole reagents via three components reaction. Neuroquantology 2021;19:115-22.
33. Aljamali NM, Jad F. Preparation, diagnosis and evaluation of cyclic-tryptophan derivatives as anti breast cancer agents. Biomed Pharmacol J 2021;14:2296.
34. Kanavy HE, Gerstenblith MR. Ultraviolet radiation and melanoma. Semin Cutan Med Surg 2001;30:222-8.
35. Ismail AS, Shukker AH, Fayad AA. Production of hydrogen and nanocarbon by catalytic decomposition of electrocracking gas over an industrial catalyst under integrated reactor conditions. Energy Proc Link 2017;141:315-31.
36. Gabar A, Maged A, Fayad AA. Synthesis and spectroscopic characterization of new heterocyclic compounds derivatied from 1-(4-aminophenyl) ethan-1-oneoxime as a starting material with evaluate their biological activity. Biochem Cell Arch 2020;20:5211-22.
37. Burnett ME, Wang SQ. Current sunscreen controversies: A critical review. Photodermatol Photoimmunol Photomed 2011;27:58-67.
38. Kütting B, Drexler H. UV-induced skin cancer at workplace and evidence-based prevention. Int Arch Occup Environ Health 2010;83:843-54.
39. Balk SJ, Council on Environmental Health; Section on Dermatology. Ultraviolet radiation: A hazard to children and adolescents. Pediatrics 2011;127:588-97.
40. Lin JS, Eder M, Weinmann S. Behavioral counseling to prevent skin cancer: A systematic review for the U.S. Preventive services task force. Ann Intern Med 2011;154:190-201.
41. Aljamali NM, Jawad AM, Kam I. Public Health in Hospitals. 1st ed. Europe: Eliva Press; 2020.
42. de Martel C, Georges D, Bray F, et.al. Global burden of cancer attributable to infections in 2018: A worldwide incidence analysis. Lancet Glob Health 2020;8:e180-90.
43. Mahdi AK, Aljamali NM. Heterocyclic-derivatives with aspirin drug (synthesis, characterization, studying of its effect on cancer cells). Int J Cell Biol Cell Proc 2020;6:30-7.
44. Wild CP, Weiderpass E, Stewart BW, editors. World Cancer Report: Cancer Research for Cancer Prevention. Lyon: International Agency for Research on Cancer; 2020.
45. Haverstick S. Patients' hand washing and reducing hospital-acquired infection. Crit Care Nurse 2017;37:e1-8.
46. Aljamali NM. Synthesis of antifungal chemical compounds from fluconazole with (pharma-chemical) studying. Res J Pharm Biol Chem Sci 2017;8:564-73.
47. Diehl JF. Food irradiation-past, present and future. Radiat Phys Chem 2002;63:211-5.
48. Aljamali NM. Designation of Macrocyclic sulfazan and Triazan as innovated compounds with their estimation in nano-activities by the scanning microscope. Int J Converg Healthc 2022;2:25-34.
49. Aljamali NM. Origination of macrocyclic formazan with macrocyclic sulfazan and Triazan as innovated compounds and compared their efficiency against breast cancer. Open Access J Biomed Sci 2022;4:383.
50. Aljamali NM. Synthesis innovative cyclic formazan compounds for the first time and evaluation of their biological activity. Int J Polymer Sci Eng 2021;7:5-14.
51. Aljamali NM. Inventing of macrocyclic formazan compounds with their evaluation in nano-behavior in the scanning microscope and chromatography. Biomed J Sci Tech Res 2022;41:32783-92.
52. Aman WA, Weber DJ. Disinfection in health care facilities: What clinicians need to know. Clin Infect Dis 2004;39:700-9.
53. Sala AA, Weber DJ, Society for Healthcare Epidemiology of America. Guideline for disinfection and sterilization cal instruments. Infect Control Hospit Epidemiol 2010;31:110-7.
54. Roth S, Feichtinger J, Hertel C. Characterization of Bacillus subtilisspore inactivation in low-pressure, low-temperature gas plasma sterilization processes. J Appl Microbiol 2010;108:521-31.

55. Kanemitsu K, Imasaka T, Ishikawa S, Kunishima H, Harigae H, Ueno K, *et al*. A comparative study of ethylene oxide gas, hydrogen peroxide gas plasma, and low-temperature steam formaldehyde sterilization. Infect Control Hosp Epidemiol 2005;26:486-9.
56. Mushtaq M, Banks CJ, Heaven S. Effectiveness of pressurised carbon dioxide for inactivation of Escherichia coli isolated from sewage sludge. Water Sci Technol 2012;65:1759-64.
57. Aljamali NM, Aliaa HM, Maryam JN. Review on analysis of breast tumor with methods of treatment. Open Access J Biomed Sci 2022;4:1580-6.
58. McDonnell G, Burke P. The challenge of prion decontamination. Clin Infect Dis 2003;36:1152-4.
59. Rogez-Kreuz C, Yousfi R, Soufflet C, Quadrio I, Yan ZX, Huyot V, *et al*. Inactivation of animal and human prions by hydrogen peroxide gas plasma sterilization. Infect Control Hosp Epidemiol 2009;30:769-77.