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Mechanistic Basis and Therapeutic Strategies in Immune Evasion in Cancer

Alireza Heidari[1,2,3,4*], Elena Locci[1,2,3] and Silvia Raymond[1,2,3]

[1]Faculty of Chemistry, California South University, 14731 Comet St. Irvine, CA 92604, USA
[2]BioSpectroscopy Core Research Laboratory, California South University, 14731 Comet St. Irvine, CA 92604, USA
[3]Cancer Research Institute (CRI), California South University, 14731 Comet St. Irvine, CA 92604, USA
[4]American International Standards Institute, Irvine, CA 3800, USA

***Corresponding Author:** Alireza Heidari, Faculty of Chemistry, California South University, 14731 Comet St. Irvine, CA 92604, USA, Email: Scholar.Researcher.Scientist@gmail.com; Alireza.Heidari@calsu.us; Central@aisi-usa.org

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Abstract

One of the most popular types of skin cancer is acral lentiginous melanoma, which usually appears as an irregular, prominent growth on the palms of the hands, feet, or under the nails. In fact, the symptoms of this cancer, which is a prominent colored spot on the skin, slowly begin to appear. In the first stage, malignant cells remain inside the tissue for months or years. The lesion then acts aggressively and appears on the skin as it exits the epidermis. Experts say this type of melanoma can grow rapidly and penetrate deep into the skin. Unlike other skin cancers that occur due to overexposure to the sun, acral melanoma has nothing to do with it. In appearance, these types of cancer spots are more than 6 mm in size and can be brown, blue-gray, black or red. Early in the onset of the disease, the melanoma may have a smooth surface, but over time it becomes thicker and has a dry, uneven surface. Bleeding and sores on the cancerous spot are also possible in some cases. Now that we know that this type of cancer is not caused by the sun's rays, then what is the reason for its occurrence? Experts say our skin has natural pigments. However, melanoma linginosis develops when some malignant pigment cells begin to proliferate in the primary layers of the epidermis. Scientists do not yet know for sure why pigment cells become malignant, but it may be rooted in genetic mutations. When a doctor diagnoses skin cancer in a person, he or she removes the cancerous spots. This process can be more complicated depending on the size of the cancer cells. If the cancer has spread to the lymph nodes, the healing process will take longer. As with other cancers, early detection of skin cancer can speed up the healing process. Therefore, after seeing any spots or colored spots on the palms of your hands, feet or under your nails, see a specialist immediately.

Keywords: Cancer; Cells; Tissues; Tumors; Prevention; Prognosis; Diagnosis; Imaging; Screening; Treatment; Management

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Introduction

The driving force behind skin cancers, such as squamous cell carcinoma, is the sun's ultraviolet (UV) light. Ultraviolet light causes mutations in skin cells, and the mutated cells proliferate before they become skin cancer. But they get help from an unlikely source. In this study, Girardy's team showed that specific immune cells and their growth factors stimulate mutated cancer progenitor cells to reproduce using genetic expression profiles, cell isolation, and preclinical models of skin cancer development in the laboratory. All of this happens microscopically, before any visible tumors appear on the skin. These are very early stages in the development of skin cancer. During exposure to ultraviolet light, skin cells stimulate other immune cells to produce growth factors, including interleukin-22, in the epidermis. Normally, interleukin-22 helps repair damaged skin, but in this example, it makes progenitor cancer cells safe to multiply. Importantly, Girardy's team discovered that all the immune cells involved in this process express a protein called RORγt. The researchers found that when they used an RORγt inhibitor on the skin surface, it greatly reduced the growth of mutated cells. We think this is the way to use similar inhibitors to prevent skin cancer in people with sun-damaged skin, especially those who are more at risk, including people with fair skin as well as those with a personal history. Or they are family skin, it opens [1-490].

Results and Discussion

Researchers have developed a two-dose cancer vaccine using Oxford vaccine technology. The cancer vaccine, when tested in mouse tumor models, increased the level of anti-tumor T cells that penetrated them and improved the immunotherapy effect of the cancer. Compared with immunotherapy alone, the combination with the vaccine showed a greater reduction in tumor size and improved survival in mice. Cancer immunotherapy transforms the patient's immune system into a tumor, leading to dramatic improvement in outcomes in some cancer patients. Anti-PD-1 immunotherapy works by removing the brakes from anti-tumor T cells to allow them to kill cancer cells. Despite this success, anti-PD-1 therapy is ineffective in most cancer patients. One reason for the poor efficacy of anti-PD-1 cancer treatment is that some patients have low levels of anti-tumor T cells. The Oxford vaccine technology used to make the famous Strazenka vaccine produces strong T + CD8 cell responses that are required for good antitumor effects. The team developed a two-dose vaccine with primary viral vectors and various boosters, one of which is identical to the Covid-19 Oxford-Astrazenka vaccine vector. In order to create a vaccine treatment that specifically targets cancer cells, the vaccine was designed to target two MAGE-type proteins found on the surface of many types of cancer cells. These two targets, called MAGE-A3 and NY-ESO-1, were previously approved by the Ludwig Institute. Preclinical trials on mouse tumor models have shown that the cancer vaccine increases the level of tumor-infiltrating CD8 + T cells and increases the response to anti-PD-1 immunotherapy. The combination vaccine and anti-PD-1 treatment resulted in a further reduction in tumor size and improved survival in mice compared with anti-PD-1 therapy alone. MAGE proteins have an advantage over other cancer antigens as vaccine targets because they are present in a wide variety of tumors. This potential advantage extends this method to people with different types of cancer. Given the importance of the target, MAGE-type antigens are not present on the surface of normal tissues, which reduces the risk of side effects from the immune system attacking healthy cells. Our cancer vaccines produce strong CD8 + T cell responses that penetrate tumors and show high potential to increase the effectiveness of immunosuppression therapy and improve outcomes for cancer patients. We combine our basic scientific knowledge of immunology and antigen detection with translational research on vaccine substrates.

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By bringing these teams together, we can address the important challenge of extending the effects of immunotherapy to the benefit of more patients.

Conclusion

There is a clear link between taking antibiotics and increasing the risk of colon cancer in the next five to ten years. This has been confirmed by researchers at Cancer Research Institute (CRI) of California South University (CSU) after studying 40,000 cases of cancer. The effect of antibiotics on the gut microbiome is thought to be behind the increased risk of cancer. The results show that there are many reasons to limit antibiotics. While antibiotic treatment is necessary in many cases and saves lives, caution should be exercised in the case of less serious illnesses that can be expected to improve anyway. "All of this is to prevent bacterial resistance, but as this study shows, it could also be because antibiotics may increase the risk of colon cancer in the future", a cancer researcher at Cancer Research Institute (CRI) of California South University (CSU). The researchers found that men and women who took antibiotics for more than six months were 17 percent more likely to develop colon cancer; Men taking antibiotics are also at risk for rectal cancer and women are more likely to develop rectal cancer. An increased risk of colon cancer was seen five to ten years after taking antibiotics. Although the increased risk was higher for those who took most antibiotics, the risk of cancer could be significantly smaller but statistically significant after a period of antibiotic use. The present study uses data from 40,000 registered patients with colorectal cancer in the United States from 2010 to 2016. These are compared with an adapted control group of 200,000 cancer-free people in the United States. Data on antibiotic use were collected from records of drugs prescribed for the period 2005-2016. To understand how to increase the risk of antibiotics, the researchers also studied a non-antibiotic antibacterial drug that is used against urinary tract infections and does not affect the microbiome. There was no difference in the incidence of colorectal cancer in people taking the drug, indicating that the effect of antibiotics on the microbiome increased the risk of cancer. While this study only covers oral antibiotics, you should be aware that studies have shown that intravenous antibiotics may affect the gut microbiota in the gut system. The mere use of antibiotics cannot be a reason for warning, the increase in risk is moderate and its effect on the absolute risk for the person is very small. The United States is also introducing routine screening for colon cancer.

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Authors' Brief Biographies

Prof. Dr. Alireza Heidari, Ph.D., D.Sc. is a Full Distinguished Professor and Academic Tenure of Chemistry and also Enrico Fermi Distinguished Chair in Molecular Spectroscopy at California South University (CSU), Irvine, California, USA. He has got his Ph.D. and D.Sc. degrees from California South University (CSU), Irvine, California, USA. Furthermore, he has double postdocs in Project Management, Oncology, Human Cancer Tissues and Synchrotron Radiation from Monash University, Melbourne, Victoria, Australia and also in Nanochemistry and Modern Molecular Electronic–Structure Computations Theory from California South

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University (CSU), Irvine, California, USA. His research interests include Biophysical Chemistry, Biomolecular Spectroscopy, Quantum Chemistry, Nanochemistry, Modern Electronic Structure Computations, Theoretical Chemistry, Mathematical Chemistry, Computational Chemistry, Vibrational Spectroscopy, Molecular Modelling, Ab initio & Density Functional Methods, Molecular Structure, Biochemistry, Molecular Simulation, Pharmaceutical Chemistry, Medicinal Chemistry, Oncology, Synchrotron Radiation, Synchrocyclotron Radiation, LASER, Anti–Cancer Nano Drugs, Nano Drugs Delivery, ATR–FTIR Spectroscopy, Raman Spectroscopy, Intelligent Molecules, Molecular Dynamics, Biosensors, Biomarkers, Molecular Diagnostics, Numerical Chemistry, Nucleic Acids, DNA/RNA Monitoring, DNA/RNA Hypermethylation & Hypomethylation, Human Cancer Tissues, Human Cancer Cells, Tumors, Cancer Tissues, Cancer Cells, etc. He has participated at more than five hundred reputed international conferences, seminars, congresses, symposiums and forums around the world as yet. Also, he possesses many published articles in Science Citation Index (SCI)/International Scientific Indexing (ISI), Medline/PubMed and Scopus Journals. It should be noted that he has visited many universities or scientific and academic research institutes in different countries such as United States, United Kingdom, Canada, Australia, New Zealand, Scotland, Ireland, Netherlands, Belgium, Denmark, Luxembourg, Romania, Greece, Russia, Estonia, Ukraine, Turkey, France, Swiss, Germany, Sweden, Norway, Italy, Austria, Czech Republic, Hungary, Poland, South Africa, Egypt, Brazil, Spain, Portugal, Mexico, Japan, Singapore, Malaysia, Indonesia, Thailand, Taiwan, Hong Kong, Philippines, South Korea, China, India, Kingdom of Saudi Arabia, Jordan, Qatar, United Arab Emirates, etc. as research fellow, sabbatical and volunteer researcher or visitor and so on heretofore. He has a history of several years of teaching for college students and various disciplines and trends in different universities. Moreover, he has been a senior advisor in various industry and factories. He is expert in many computer programs and programming languages. Hitherto, he has authored more than twenty books and book chapters in different fields of Chemistry. Syne, he has been awarded more than one thousand reputed international awards, prizes, scholarships and honors. Heretofore, he has multiple editorial duties in many reputed international and peer–reviewed journals, books and publishers. Hitherward, he is a member of more than five hundred reputed international academic–scientific–research institutes around the world. It should be noted that he is currently the President of the American International Standards Institute (AISI), Irvine, California, USA and also Head of Cancer Research Institute (CRI) and Director of the BioSpectroscopy Core Research Laboratory at California South University (CSU), Irvine, California, USA.

Elena Locci is a Ph.D. Candidate under the Supervision of Professor Alireza Heidari at Cancer Research Institute (CRI) and BioSpectroscopy Core Research Laboratory at California South University (CSU), Irvine, California, USA.

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Dr. Silvia Raymond, Ph.D., D.Sc. is the current Junior Postdoctoral Research Fellows under the Supervision of Professor Alireza Heidari at Cancer Research Institute (CRI) and BioSpectroscopy Core Research Laboratory at California South University (CSU), Irvine, California, USA.