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Research Article

'GENOME MODULATING INTRACELLULAR ACQUIRED IMMUNITY' (GMIAI) IN CANCER CELLS MIGHT BE CANCER'S DEFENCE MECHANISM

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Abstract

The 3D genome structure alteration and 'specific antidrug group of protein combinations (SAGPC)'production are two main contents in my hypothesis for chloroquine resistance in malaria parasites, later, it was expanded to unify the mechanisms of drug resistance in cellular pathogens across cancer cells, bacteria, protozoa, and fungi. The foundation of this hypothesis is based on "genome modulating intracellular acquired immunity" (GMIAI) in cellular pathogens including cancer cells. Now I want to expand this hypothesis to cancer cells because cancer cells face many hostile molecules that are drugs, hostile molecules from immune system or immunotherapy and environment. Thus, GMIAI in cancer might produce various 'antibodies' for fighting various hostile molecules. The 'antibodies' here are 'specific anti-hostile-molecule group of protein combinations' (SAGPCs) that replaces original SAGPC that is only for antidrug. The cells in multicellular organism don't have good function of GMIAI because they are protected by immune system, while cellular pathogens belong to unicellular organism and no immune system for protecting them. Simply, proteins in multicellular-organism cells are fully controlled by multicellular organism and might have little tendency for making SAGPCs, while proteins in unicellular-organism cells like cancer cells could have active tendency for producing various SAGPCs to specifically fighting various hostile molecules for protecting cancer cells. Therefore, GMIAI in cancer cells might be the cancer's defence mechanism. Majority of cancer patients died of metastasis, which might be closely linked to this cancer's defence. In this paper, I would try to present in detail about GMIAI in cancer cells and its linkage to metastasis.

Keywords: Cancer's defence mechanism**, '**Genome modulating intracellular acquired immunity'(GMIAI), 'Specific anti-hostile-molecule group of protein combinations (SAGPC)'**,** Cancer metastasis.

INTRODUCTION

Cancer remains on top death list globally, over 20 million cancer cases are diagnosed annually worldwide, causing 10 million deaths [1]. In medical practice, metastasis account for 90% death, becoming the leading causes of cancer deaths in human [2]. The evolution of metastasis in humans is not well understood and the reason is still debating scientifically [3]. Some researchers think that cancer cells are heterogeneous and contain clones that have metastatic properties, which could be selected out. The metastatic tumour is found in distant organs because they have different microenvironment that can influence the growth, invasion, and response to chemotherapy [4]. Obviously some cancer cells from primary cancer spread to distant organ and start colonization there, forming a metastatic tumour that damage the function of vital distant organs. Why doesn't cancer love hometown (primary cancer location) and must move to new home (metastatic tumour in distant organs)? Why small number of cancer cells in blood and lymph fluid are not killed by immune defence? Why metastatic tumour is more difficult to treat than primary tumour?

To answer these questions, I think that 'genome modulating intracellular acquired immunity' (GMIAI) in cancer cells is vital important in protection of cancer cells. The concept of GMIAI was first proposed in my hypothesis about 3D genome structure alteration and 'specific antidrug group of protein combinations' (SAGPC) production, which attempts to unify drug-resistance mechanisms of cellular pathogens across cancer cells, bacteria, protozoa, and fungi [5]. In this paper, I present in detail about cancer cell's GMIAI and its linkage to cancer metastasis.

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'GENOME MODULATING INTRACELLULAR ACQUIRED IMMUNITY' (GMIAI) IN CANCER CELLS

In 2023, the concept of 'genome modulating intracellular acquired immunity' (GMIAI) was first proposed in my hypothesis that attempts to unify all mechanism in cellular pathogens across cancer cells, bacteria, protozoa, and fungi [5]. Now I want to expand this hypothesis to cancer cells because cancer cells face many hostile molecules that are drugs, hostile molecules from immune system or immunotherapy and environment. Thus, GMIAI in cancer cells might produce various 'antibodies' for specifically fighting various hostile molecules. The 'antibodies' here are 'specific anti-hostile-molecule group of protein combinations' (SAGPCs) that replaces original SAGPC that is only for antidrug. Theoretically, the GMIAI should exist in all eukaryotic cells, but the cells in multicellular organism don't have good function of GMIAI because they are usually protected by immune system, while cellular pathogens belong to unicellular organism and no immune system for protecting them. Simply, proteins in multicellular-organism cells are fully controlled by multicellular organism and might have little tendency to produce SAGPCs, while as a unicellular organism. cancer cells have strong tendency to produce SAGPCs for protecting themselves. Therefore, GMIAI in cancer cells could be cancer's defence mechanism. Together, cancer cells might fight all hostile molecules through this defence system. Each

SAGPC fights one hostile molecule specifically and many proteins are shared by some SAGPCs.

PROTECTION OF CANCER CELLS BY GMIAI AND CANCER METASTASIS

Cancer cells in human face the harsh surround by many hostile molecules such as drug therapy (DT), hostile molecules from immune system or immunotherapy (HMI) and hostile molecules from environment (HME). Here I use DT, HMI and HME just for representing all hostile molecules, which is simple for explanation. Therefore, to protect cancer, GMIAI in cancer cells will produce virous SAGPCs in each of anti-DT, anti-HMI and anti-HME 'antibodies' after cancer's long-time exposure to drug and hostile molecules from immune system or immunotherapy and environment. One SAGPC means one 'antibody'. There are many SAGPCs in anti-DT, anti-HMI and anti-HME 'antibodies', each of SAGPCs specifically fighting one hostile molecule. Some proteins are shared by some SAGPCs. Conjecturally, I think that GMIAI in cellular pathogens might have memory function because alteration of 3D genome structure could leave trace on chromatin fiber [6]. Cancer cells with anti-drug, anti-HMI and anti-HME properties will become a bit unbeatable, this is the reason why they can travel through bloodstream and lymph fluid and eventually grow in distant organs. Cancer don't love hometown (primary location) because they are hungry without enough nutrition and suffering harsh surround by hostile molecules from immune system or immunotherapy and environment, which is the reason why they like to live in new home (metastasis in distant organs) because they have enough food and no harsher surround there. The whole story is concluded and shown in Figure 1

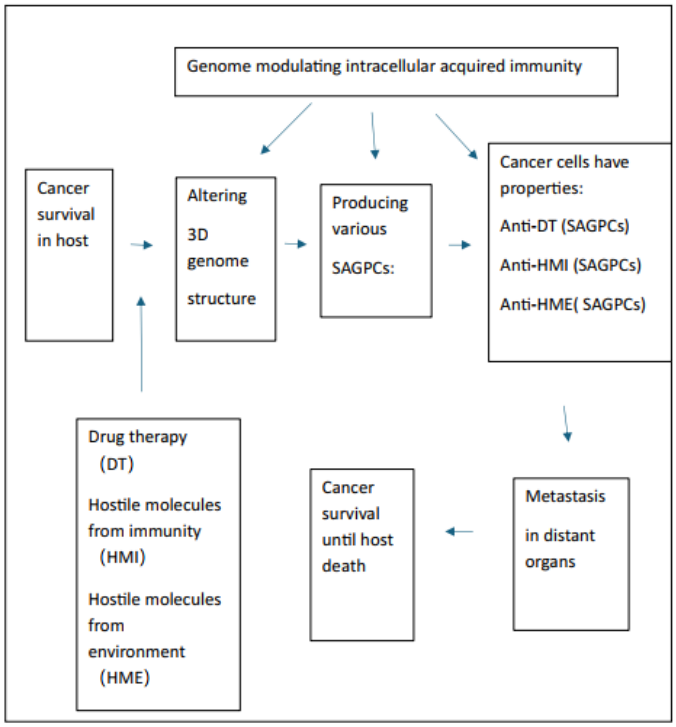


Figure 1. 'Genome modulating intracellular acquired immunity' (GMIAI) in cancer cells and its function in completion of cancer metastasis that causes majority deaths of cancer patients

There are many SAGPCs produced by cancer's GMIAI system, but it is difficult to isolate a single SAGPC from cancer cells because proteins are mixed up together to form a 'protein soup'. However, to validate existence of SAGPCs, comparative transcriptome analysis could be used [7]. First, the cancer cells are divided into 3 groups, group A, group B, and group C. Making group A become resistance to a drug (D1), making group B become resistance to another drug (D2), and making group C become resistance to two drugs (D1 and D2). Second, the drug-resistant 3 groups are subjected to comparative transcriptome analysis, the differential proteins in group A and group B are two SAGPCs, respectively, and the differential proteins in group C are mixed SAGPCs. Comparison of the 3 groups' differential proteins, the proteins that are shared in two SAGPCs could be confirmed. Together, this approach will be able to validate existence of SAGPCs, which further proves existence of GMIAI system.

CONCLUSION

This paper presents a hypothesis, which might inspire researchers to find the solution for dealing with metastasis and evaded immunotherapy in cancer patients and help them living longer. The GMIAI in cellular pathogens including cancer cells might be a new systematic defence process that I proposed, which might not be recognised by mainstream medicine. But I just hope that it could be integrated into modern medicine eventually. I believe that there are many good idea or insights in articles published in low-ranking open access journals. Some persons call these journals predators, against whom I am because they want to kill science freedom and just serve for high-ranking journals. Honestly, without these journals, my voice will not be heard. Many mainstream researchers read articles in these journals, but don't cite them because they may get inspiration that make them do the similar work that could be published in high-ranking journals. This is darkness in scientific community. I have published few papers with insightful idea, for example, in1986, I published abnormal chromatin configuration causing cancer formation [8]; in 2020, I published a novel antiviral theory [9][10]; and in 2023, I published the unified mechanism of drug resistance in cellular pathogens [5]. But no articles worldwide have cited them till present, which make me disappointed about the darkness in scientific community. Undoubtedly, I foresee that this paper might face the same bad future.

CONFLICTS OF INTEREST

The author declares no conflicts of interest.

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