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Insights into Potential Interactions Among Antimicrobial-Like Amphipathic Peptides, Tumor Stem Cells and Cancer Recurrence: A Commentary and Theoretical Prospectus

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Abstract

The rates of cancer recurrence many years after a "cancer-free" status can be a discouraging revelation for patients. Many, if not most cancers, can return and regrow in patients after many cancer-free years. However, the use of antimicrobial peptides in cancer therapeutics has slowly increased in past years and would suggest a promising future. The present report addresses the interaction that appears to exist among:

i. Antimicrobial-like amphipathic peptides,

ii. Cancer stem cells, and

iii. The growth recurrent cancers; taken together, these elements could have promising future implications.

Among the many advantages of using antimicrobial-like peptides, these amphipathic peptides display remarkable cancer cell targeting properties and are capable of transporting and delivering chemo-drug payloads into malignant cells. In addition, the cancer cell targeting abilities of peptides have been found to be dependent on a net negative surface charge displayed on the cancer cell bilayer surface membrane. Thus, one could postulate that the destruction of tumor cells, together with cancer stem cells, could possibly alter and reduce the recurrence rates of cancer patients in future years to come.

Keywords: Alpha-Fetoprotein; Antimicrobial Peptides; Cancer; Recurrence; Stem Cells; Chromosome Instability; Amphipathicity; Cell Membranes; Electrical Change

Abbreviations: AFP: Alpha-Feto Protein; AMLPs: Antimicrobial-Like Peptides; TRP: Transient Receptor Potential; CSC: Cancer stem cells

Introduction

Antimicrobial-like Amphipathic Peptides

The antimicrobial-like peptides (AMLPs) are naturally occurring peptides found in a multitude of organisms [1]. The original AMLPs were found to be broad spectrum antibiotic agents that specifically killed gram-positive and gram-negative bacteria and could further attack enveloped viruses and transformed cancer cells in mammals including man [2,3]. The AMLPs can create pores and/or transmembrane channels within the surface bilayer of cell membranes to translocate into the cell cytoplasmic interior [2].

These newly formed channels are like the already in-place Transient Receptor Potential (TRP) channels, and the two together are associated with other receptors, capable of interacting with invasive AMLPs. The function of the AMLPs is to destabilize, disrupt, and perturb the bilayer membrane to allow cell penetration and entrance into the cancer cell cytoplasmic interior which can subsequently affect other intracellular transduction pathways.

The AMLPs can extend in length from 12 to 50 amino acids, contain two or more arginines, lysines, or histidines, and display several hydrophobic and polar amino acids; overall, these entities are largely zwitterionic and amphipathic peptides [4,5]. The secondary structure of such peptides can consist of alpha-helices,

beta-sheets, hairpin loops, and usually contain one or more disulfide bridge linkages [6]. Some AMLPs can also contain random coil (disordered) structures as part of their secondary structure.

The main targeting objective of the AMLPs is the cell surface bilayer membrane in which these peptides are capable of disrupting and destabilizing the bilayer leaflet of the cell membrane [7,8]. In this manner, the AMLPs can permealize (penetrate) into the cell membrane bilayer and form a pore and/or a transmembrane channel to gain cell entry. Such peptides are attracted to and bind to a net negative surface charge on the cell membrane by means of an electrical attraction to the alpha-helical content, amino acid composition, and amphipathic nature of the peptide [8].

It is of interest that a net negative charge on the cell surface membrane is displayed only on bacteria, stem cells, and transformed cancer cells. In contrast, normal non-cancer mammalian cells display only a net positive cell surface membrane charge [9,10]. The net negative charge on cancer and stem cells results from undergoing a phospholipid bilayer flip, in which the innermost negative-charged phospholipid head groups emerge (flip) to the outer bilayer of the cell membrane, while the positive charged phospholipids retreat to the inner membrane leaflet bilayer location [11].

Such bilayer-altered cancer cells subsequently exhibit a net negative cell surface membrane charge. Such a negative charged surface membrane allows the AMLPs to home in on and target only cancer cells, but not normal non-malignant cells [12]. Is it possible that AMLPs presently exist in the toolbox of cancer research biologists? The answer is affirmative in that most AMLPs possess these traits especially in reference to the Growth Inhibitory Peptide, which is derived from the full-length alpha-fetoprotein (AFP) polypeptide. This AFP-derived peptide has long been researched and reported in biomedical literature and is capable of confronting, attacking, and binding to cancer cells in both in vivo and in vitro studies [13-15] (Table 1).

Table 1: The Antimicrobial Peptides are listed according to their biochemical and biophysical characteristics, traits, and properties.

Characteristics, Traits, and Properties	Antimicrobial Peptides (AMP)	References Cited
1) Cell membrane penetration effects	Forms transmembrane pores and/or channels, stabilizes the cell membrane potential	01-Mar
2) Cell method of internalization	Transmembrane channel passage, channel receptor endocytosis	7
3) Cell-specific targeting	Microbial cell wall/membrane, plasm membranes of vertebrate (mammals), transformed cancer cells and bacteria	8
4) Cell cargo delivery vehicles	Many have cargo delivery capability, binds metals, dimerizes with peptides and proteins	7, 8
5) Cell toxicity	Cytostatic, Cytolytic	3
6) Traits and characteristics of amino acids	Largely amphipathic, contains some positive and negative charged AAs and hydrophobic AAs	9
7) Amino acid length	12-50 AAs	10
8) Peptide secondary structure	Displays some alpha-helix, beta sheets, and beta hairpin loops	11
9) Effect on Host Immunity	Promotes and regulates the innate response of host organism, initiates chemokine immunomodulation	12, 13
10) Examples of peptides in nature and/or synthesized	a) Amphibian-H5 b) Human dermcidin c) Human defensins d) Cecropins from insects e) Magainin and bombinin from amphibians f) Indolicidin from cows g) Prophenin from pigs h) Tachyplesin from horseshoe crabs	13-15

Cancer Stem Cells

Cancer stem cells (CSC) are a small population of cells (2-3%) that reside in tumors and which are self-renewing, transformational, and multipotent. The CSCs are most similar and resemble the stem cells located in hematopoietic tissues; although such stem cells are slow growing, they retain strong long-term stem-

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to-tumor cell-transforming capabilities [16]. The CSCs have been detected and identified in multiple cancer cell types, including breast, brain, intestinal, gastric, liver, and many others [17]. It has been found and reported that such stem cells appear to be the source of tumor recurrence in patients many years after successful medical treatments; thus, even though many patients have been declared "cancer-free" at a previous time, the malignant tumor may return [18]. Thus, CSCs are a specific cell phenotype that can eventually progress to tumor transformation, regrowth, invasiveness, and recurrence many years later.

In lieu of the above discussion, many non-cancer organs have a small reserve of stem cells to replace worn out cells; in addition, many stem cells can further be stored in fatty tissues in other part of the body. In fact, a subset of mesenchymal derived stem cell population (1-3% of total cells) has further been detected in the abdominal fatty storage tissues of the body. In summary, the present treatise should be deemed significant in attempting to explore, study, and understand the main activators, mediators, and regulatory cell mechanisms involved in the historical development of cancer stem cells.

The Origin of Cancer Stem Cells

The origin of CSCs is still in debate and not fully understood. Thus, the precise origin of cancer stem cells has not been definitively established with complete certainty. Nonetheless, three proposed sources of CSCs are thought to be derived from: 1) normal body stem cells; 2) genetically altered adult cells; and 3) transformed fusion cells [19]. However, it appears to be generally accepted in the medical community that most CSCs originate from normal body stem cells and/or precursor cells, which have undergone multiple gene mutations.

Several opinions have been forwarded regarding the traits of CSCs, but one common characteristic appears to be that of genetic instability which is the fundamental basis for the transformation of stem cells or progenitor cells into CSCs [16,20]. One such hypothesis states that normal stem cells have long lifetimes, allowing for accumulation of numerous oncogenes and/or tumor suppressor gene mutations that produce genomic changes, especially the transformation of stem cells into tumor cells [21]. Such events could originate at the chromosome and/or molecular levels in which normal stem cells could differentiate into CSCs; these then exhibit different genotypes and phenotypes from those of normal body stem cells.

The CSCs can acquire increased oncogenes such as C-Myc, RAS, and Notch which manifest the traits of slow-growing and self-renewal abilities, shorter cell cycles, and adaptation to hypoxia environments for ease of cell transformations and transitions [21]. Stem cells with tumor-initiating abilities often display certain cell surface biomarkers on their cell membranes such as CD34+ and CD38+immune-associated markers. The precise origin of CSCs from adult and cell fusions are less understood and will not be further addressed in the present report.

Stem Cells Display a Net Negative Charged Cell Membrane

Stem cells, like all cells, are composed of a cell membrane lipid bilayer composed of two layers of phospholipids with cholesterol interspersed between the two bilayers. The presence of cholesterol ensures that the membrane fluidity (ability to shift movements within the cell membrane) is maintained within the cell membrane. In normal non-malignant cells, the outer layer of the cell membrane is composed of positive-charged phospholipid head groups, while the inner bilayer is composed of negative-charged phospholipid headgroups [22]. Certain cells can undergo a lipid flip in the membrane bilayer which transfers the inside negative charged layer to the outside bilayer leaflet of the cell membrane and shifts the positive charge phospholipids back to the inner membrane leaflet [11]. In this manner, while the outer cell membrane of normal cells displays a net positive surface charge, the stem cells in contrast exhibit a net negative charge on their outer cell surface membrane bilayer. In summation, the net negative surface charge of stem cells is a direct result of the transfer or shift of the negative phospholipid headgroups to the outermost membrane bilayer [11,23].

In contrast, the normal cells of the body carry a net positive surface charge on their outer bilayer due to their positive charged phospholipids. The negative charge of stem cells resulted from the zeta potential that is typically measured as negative (range from -80mV to -25m voltage) depending on the cell type and interstitial space conditions [22]. The cell membrane potential refers to the electrical charge differential across the cell membrane, which is involved in the downstream transmission of transduction signals within cells [24,25]. It is the anionic phospholipids that confer a net negative charge on the outermost bilayer cell membranes of stem cells. Thus, it is the difference in ion concentrations in the cell interior and exterior that gives rise to the electrical potential across the cell membrane.

Cancer Recurrence Rates

The thought of cancer "return" or "recurrence," after several years in prior cancer-free patients, produces a significant fear in both patients and in their caregivers [26]. Oncologists are encouraged to discuss cancer recurrence rates considering symptoms and practices with their past and present patients; these discussions are regarding the rationale behind various follow-up procedures which can vary widely in patients with different types of cancer. Such procedures are based on disease stage, tumor pathology, patients' genetic background, and various patient prior treatments.

However, it is important to view recurrence rates only as statistical estimates due to the different cancer cell types, ongoing new developments of anti-cancer therapies, and differences between individual-recovering cancer patients [27]. Summaries of

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the different recurrence rates in adults are listed in Table 2 presented as three different categories, namely: A) Cancers with high recurrence rates; B) Cancers with medium recurrence rates, and C) Cancers with low recurrence rates. The childhood cancer recurrence rates are further listed in Table 3.

Table 2: Recurrence Rates in Various Adult Cancers.

*This table was derived, reconfigured, and re-designed from: The Cancer Therapy Advisor, "Current Recurrence Statistics," Nov, 30, 2018 by: Andrea S. Blevine Primeau, Ph.D., MBA

A) Cancers with Low Recurrence Rates (1-33%)	
Name of Cancer Type	**Percent Recurrent Rate (%)**
1. Breast Cancer	31%
2. Colorectal Cancer	17%
3. Head and Neck Cancer	22%
4. Hodgkin's Lymphoma	20%
5. Acute Lymphoblast Leukemia	20%
6. Non-small Cell Lung Carcinoma	27%
7. Acute Myeloid Leukemia	29%
8. Osteosarcoma (non-metastatic)	12%
9. Prostate Cancer	24%
10. Thyroid Carcinoma	30%
11. Thyroid (postsurgical) Carcinoma	14%
B) Cancers with Medium Recurrence Rates (34-66%)	
1. Bladder Cancer	50%
2. Kidney Cancer	49%
3. Lymphoma (diffuse large B-cell)	35%
4. Melanoma (non-metastatic)	41%
5. Osteosarcoma (metastatic)	45%
6. Pancreatic Cancer	46%
7. Prostate Cancer	48%
8. Soft Tissue Sarcoma	50%
C) Cancer with High Recurrence Rates (67-100%)	
1. Glioblastoma Brain Cancer	100%
2. Lymphoma (peripheral T-cell cancer)	75%
3. Melanoma (metastatic)	87%
4. Ovarian Cancer	85%
5. Soft Tissue Sarcoma (Advanced Disease)	100%

Table 3: Recurrence Rates of Various Childhood Cancers.

*See Table-2 for reference citation.

Types of Childhood Cancers	Tissues/Organs Affected	Symptoms, Characteristics and Traits	Percent Cancer Recurrence Rates
1) Wilms Tumor	Kidney cells and tissues	Fever, nausea, loss of appetite, shortness of breath, constipation, blood in urine	15-20%
2) Leukemia	Blood cells	Fever, fatigue, frequent infections, shortness of breath, pale skin, bone/joint pain, easy bleeding/ bruising	20-24%
3) Rhabdomyosarcoma	Soft tissue, muscles	Pain, swelling, bleeding, headaches, bone pain, eye issues, persistent cough, weakness	50-70%

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4) Germ cell tumors	Reproductive organs	Pelvic discomfort, swollen abdomen, abdominal pain, nausea	30%
5) Non-Hodgkin's Lymphoma	Lymph nodes	Painless, swollen lymph nodes, abdominal pain, chest pain, difficulty breathing, fatigue, fever	30%
6) Retinoblastoma	Retina (eye)	Leukocoria, strabismus, poor vision, eye pain, redness, teary eyes	6-45%
7) Acute Lymphoblastic leukemia	Blood cells, specifically lymphocytes	Fatigue, fever, easy bleeding and bruising	10-20%
8) Hepatoblastoma	Liver, hepatocytes stromal cells	Abdominal pain, noticeable lump in abdomen, weight loss	20%
9) Neuroblastoma	Nerve tissue (neurons)	Swelling in neck, chest, and abdomen, bulging eyes, bone pain, weakness in extremities, paralysis, weight loss	50-60%
10) Brain tumors and Central Nervous System	Brain and brain stem cells	Headaches, seizures, difficulty thinking, behavior changes, loss of balance, vision changes, hearing loss	90%
11) Atypical Teratoid	Brain and spinal cord	Morning headaches, vomiting, changes in activity levels, nausea, fatigue, trouble with balance , seizures	18%
12) Rhabdoid Tumor	Kidney, soft tissue, central nervous system	Blood in urine, nausea, vomiting, swollen lymph nodes, irritability, decreased appetite, fatigue	20-30%
13) Osteo-Sarcoma	Bone	Bone pain, swelling and redness at site, limited movement	80%
14) Glioblastoma	Brain	Headaches, nausea, vomiting, blurred vision, seizures	75-80%

Conclusion and Future Prospects

It can readily be ascertained from the above discussion that the interactions and interplay among the three discussed elements are important. These elements are namely, 1) the AMLPs, 2) cancer stem cells, and 3) cancer reoccurrence rate statistics; knowledge of the three could suggest future encouraging therapeutic possibilities. For example, one could postulate and/or propose that the return of recurring cancers might be preventable under certain combinations of treatment conditions. The encouraging claim by oncologist that a patient is declared cancer-free, following extensive radiation and chemotherapeutic treatments, can eventually be short-lived in the patients future years to come.

Cancer stem cells are very slowly growing and may take many years to manifest and regrow into a newly detectable tumor mass. As shown above, most if not all, tumors surveyed displayed a new cancer regrowth recurrence [16]. However, with the utilization of AMLPs as a therapeutic treatment option, such peptides might specifically be able to target and home onto cancer cells and CSCs while bypassing normal non-malignant dividing body cells. Present chemotherapeutic drugs can home onto normal mitotic dividing body cells together with the cancer mitotic dividing cells, without discrimination between the two different cell types.

In addition, the above discussion further revealed that cancer stem cells display a net negative surface membrane charge, making such cells further vulnerable to AMLP therapeutic treatments. These elements have the potential to simultaneously destroy both cancer cell and cancer stem cell populations. In theory, one could propose that such AMLP treatments could simultaneously destroy both cancer cells and cancer stem cells with the same peptide therapeutic regimen. Such an action could possibly reduce the chance of cancer stem cell transforming into cancer cells and might decrease the chance of both cancer regrowth and recurrence in future years.

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Conflicts of Interest

The author declares there are no known conflicts of interest in the preparation of this manuscript.

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