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Editorial
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Major Recent Developments in Cancer Treatment

Alain L Fymat*

International Institute of Medicine & Science, California, USA

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***Corresponding author:** Alain L Fymat, International Institute of Medicine & Science, California, USA

Editorial

Cancer is possibly the oldest disease afflicting humanity for the past 4,000 years. It was hidden from view and misinterpreted because of prevailing other diseases and the relative shortness of the human's lifespan. With the victory over a plethora of such diseases and humanity's increasing longevity, cancer has emerged in full force during approximately the last two centuries and will, unfortunately, still be with us for the remainder of humanity's existence. According to the World Health Organization, "*cancer is the second leading cause of death globally, accounting for an estimated 9.6 million deaths, or one in six deaths … The cancer burden continues to grow globally, exerting tremendous physical, emotional, and financial strain on individuals, families, communities, and health systems*". For its part, the American Cancer Society has repertoried 72 types of cancer and estimated that by the end of 2015, there were approximately 1.66 million new cancer cases diagnosed in the country (this figure would increase once the global statistics are taken into consideration). Based on model-based projections for 2021, it estimated the numbers would still increase to 1,898,160 new cases and 608,570 deaths. But why hasn't cancer been cured despite a four-decade "war" against the disease and the expenditure of hundreds of billions of dollars worldwide? It is essentially because of our incomplete understanding of the basic underlying molecular mechanisms that drive it.

Cancer is not a single disease. It is a multiplicity of diseases caused by the uncontrolled growth of a single cell unleashed by mutations. Cancer cells are better versions of normal cells in terms of their growth, spread, repair ability, and longevity. We naively thought we could defeat them by preventing or even eliminating the initial occurrence of mutations without hopefully impacting normal cell growth. Unfortunately, this view did not consider the pernicious genetic intertwining of normal and cancerous growths wherein cancer is braided in our genome. Thus, we can rid ourselves of cancer only in as much as we can rid ourselves of the processes in our physiology that depend on growth – aging, regeneration, healing, reproduction. Woven into our genome. the mutated genes are but distorted versions of the normal ones; they are braided together and unbraiding them continues to be the most formidable undertaking. Fortunately, most cancer cases are due to environmental risk factors, many of which (but not all) being controllable lifestyle choices and, thus, preventable. It has been suggested that more than 30% of cancer deaths could be prevented by avoiding risk factors including: tobacco, overweight, obesity, insufficient or/and inappropriate diet, physical inactivity, alcohol, transmitted infections, and air pollution. But not all environmental causes could be controlled (e.g., naturally occurring electromagnetic background radiation).

Normally, cell division is controlled by the balancing of complementary growth and inhibition factors after which they are unable to migrate to other organs and die. When these antagonistic signals are impaired, no longer operate, or are bypassed, new genetic mutations take place, grow, escape, get transported in the blood stream, colonize distant areas, and metastasize. This development is governed by the individual's inherited tendency and environmental exposure. No less than eleven hypotheses and theories of cancer (elements of which remain nonetheless valuable) have been advanced over the years, including blood suppuration, somatic mutation, viral, retroviral, infectious mononucleosis, endogenous proto-oncogene, two-hit, inflammation, angiogenesis, hormone therapy, and immunotherapy (whether natural or synthetic). Some of the major recent developments in cancer treatment include:

Innate immunotherapy with neutrophil-mediated drug delivery for the suppression of postoperative malignant glioma recurrence. Neutrophils mediate the delivery of anti-cancer drugs. They slow tumor growth without inhibiting or preventing their

regrowth, but significantly improve survival rates. The technique can theoretically be used in inflammation-mediated disorders other than cancers and any other diseases that naturally attract neutrophils [1].

Synthetic immunotherapy using either chimeric antigen receptor T-cells or programmed-death inhibitors. In this elegant and appealing approach, the immune system is stimulated rather than targeting the cancer itself. Further, their task completed, the engineered cells remain in the body, offering future protection ("cell memory"). *Science* magazine declared it the year 2013 breakthrough! Unfortunately, today's immunotherapies do not help everyone (e.g., the odds remain long for patients with metastatic cancer) and biomarkers that might offer answers remain to be designed as well as experimenting with ways to make therapies more potent. Nonetheless, even cancers impervious to the new drugs (3%- 4%) could be treated if those malignancies have the right error-riddled DNA signature.

DNA Origami/Trojan technique to foil drug resistance in solid tumors

In its refined version, the genome of a common bacteriophage and synthetic strands that were designed to fold up its DNA are encapsulated and do not encode any proteins or do any of the normal DNA functions. Potentially, the technique should work on most any form of drug-resistant cancer.

Enzyme mnk-2 conversion to overcome drug resistance in breast, lung, and colon cancers

The enzyme mnk-2 is binary with a "normal" form that inhibits cancer development and an "abnormal" form that promotes it. The balance between these two forms will determine whether the cancer is arrested or promoted. To overcome drug resistance, molecules have been developed that can convert the abnormal to the normal form of *mnk-2*. The underlying mechanism elucidates how cancer cells eliminate the anti-cancer form and provides a means to reverse it.

Antiangiogenesis to cut-off the blood vessels alimenting the cancer cells

Cancer cells hijack and feed off blood vessels. In the brain, this results in a weakened blood-brain barrier and this may provide one reason for the rapid spread of glioblastomas. This observation may lead to new ways to kill brain tumors using the barrier's weakness to get targeted drugs into the brain during the early stages of the cancer. If the above findings hold true in humans, treatment with anti-invasive agents might be beneficial in newly diagnosed glioblastoma patients.

Self-eradication of cancer during meiosis

During anaphase in mitosis, an "inherent death mechanism" can self-eradicate duplicating cancer cells without impairing healthy cells in both normally and rapidly proliferating human cancer cells. The faster cancer cells proliferate, the faster and more efficiently they will be eradicated. The mechanism involves the modification of specific proteins that affect the construction and stability of the spindle, a newly discovered mechanism that can arrest cancer cells from dividing and multiplying, thus stopping cancer progression in its track. It may be suitable for treating aggressive cancers that are not responsive to traditional chemotherapy. A variety of drugs that also modify these specific proteins could be developed.

Combating inflammation to limit tumor invasion, progression, and metastasis

Many malignancies arise in areas of chronic inflammation and inadequate resolution of inflammation could have a major role in tumor invasion, progression, and metastasis although it may not play a role in oncogenesis. Inflammation is of pathophysiological relevance in lung cancer in that chronic bronchitis, triggered by asbestos, silica, smoking, and other external inhaled toxins, results in a persistent inflammatory response. Inflammation in the tumor microenvironment mediated by interleukin IL-1β has a major role in cancer invasiveness, progression, and metastasis and must be combated. Coupled with the monoclonal antibody Canakinumab, it has led to associated reduced incidences of fatal cancer, lung cancer, and fatal lung cancer.

Electropermeabilizing the cancer cell membrane to deliver drugs to the cell's interior

Based on the local application of short and intense electric pulses that transiently permeabilize the cell membrane, electrochemotherapy delivers non-permeant drugs or low-permeant drugs to the cell's interior. The technique has been widely used for cutaneous and subcutaneous tumors or their metastases, including adenocarcinoma, basal and squamous cell carcinoma, melanoma, and Kaposi sarcoma.

Employing nanochemotherapy to deliver nanoparticles encapsulating cytotoxic drugs to tumors

High drug levels can be delivered in several situations. The technology has numerous clinical advantages: the nanoparticles circulate throughout the bloodstream without being attacked by the immune system, preferentially bind to damaged blood vessels and certain pathogens, are non-toxic, can be safely metabolized by the body; and can be packed with many small drug molecules that diffuse out of the core and onto their targets.

Additionally, other cancer theories and therapies continue to be proposed. In addition, with the momentous advances of the Human Genome Project and its sequel the Human Cancer Genome Project, we have now come to the realization that understanding more intimately the deep biology of cancer, gene by gene, pathway by pathway, will direct us into the right direction

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for cancer therapeutics. A second direction would be to integrate our understanding of aberrant genes and pathways to explain the behavior of cancer. A third and last direction would obviously be cancer prevention, or at least prevention or/and minimization of those epigenetic and ecogenetic factors that may trigger the expression of cancer. This would lead to a personalized approach to cancer treatment along the lines advocated for a new paradigm in medicine and health care, namely, personalized medicine.

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