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Opinion

Cancer Patients' Lives Matter

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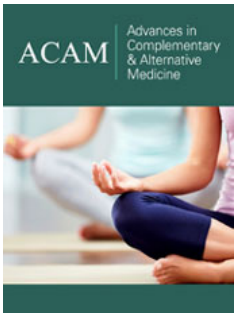

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Cancer Mortalities Remain at All Time High

Cancer mortalities are on the very top in most industrialized nations for a very long time. A notable exception is the USA. The cancer mortality of USA is second to cardiovascular diseases. The cancer mortality of China was the second to infectious diseases 50 years ago but is now surged to the first place. These mean that the health profession did not make a good progress to save the lives of cancer patients in the past. Cancer therapy is dominated by cytotoxic agents in the past. It is long overdue to deliberate cancer therapy employing toxic agents. Cytotoxic chemotherapy was a tragic byproduct of the World War II. Toxic sulfur mustard gas bomb was a weapon during the war. Victims of sulfur mustard gas all showed deficiency of lymphocytes. That finding inspired oncologists to employ toxic chemicals to treat leukemia patients. Cytotoxic agents became the dominant modality of cancer therapy, and the disappearance of tumor size became the standard criterion for the evaluation of therapeutic efficacy. Cytotoxic therapy was recommended to President Nixon to declare war on cancer in 1971 [1], which, however, failed to win the battle during the five years of intensive presidential support, and in the following 46 years of exclusive support from medical resource allocated to combat cancer. It is a shame that the health profession, which used to have the collection of very bright brains, fail to achieve a relatively easy presidential project than other much more difficult presidential Manhattan project and Apollo project. The failure to win the war on cancer is because the cancer establishments were trapped in an unwinnable modality of cancer therapy employing cytotoxic agents. Cytotoxic agents are actually inappropriate for cancer therapy [2]. Cancer arises as a consequence of wound not healing properly due to the collapse of chemo-surveillance [2-4]. Therefore, cytotoxic agents are contraindication on cancer therapy. They create more wounds to aggravate the already bad situation caused by wound not healing well. Their inability to eradicate cancer stem cells (CSCs), which are protected by a drug resistance mechanism to exclude toxic chemicals, and their contribution to further damage the functionality of chemo-surveillance lay the ground for inevitable recurrence and fatality. So even the few cancer patients fortunate to achieve complete remission undergoing therapy with cytotoxic agents are eventually succumbed to recurrence. That is why cancer mortalities remain at all time high. Cytotoxic agents can only benefit a very few early stage cancer patients whose functionality of chemo-surveillance is not fatally damaged in the process of treatment, which is restored to the functional state to subdue surviving CSCs. The majority of cancer patients are either succumbed to the adverse effects of cytotoxic agents or to the recurrence. We cannot unintentionally keep on killing cancer patients with cytotoxic agents. It is high time to turn to treatment modalities that can save the lives of cancer patients.

Destabilization of Abnormal Methylation Enzymes (MEs) as the Nature's Choice to Win the War on Cancer

Chemo-surveillance was a hypothesis brought up by Liau et al. [5] as a natural defense mechanism against cancer. The hypothesis was based on the observation that healthy

people were able to maintain a steady level of metabolites active as differentiation inducers (DIs) and differentiation helper inducers (DHIs). DIs and DHIs are metabolites that can modulate differentiation capability of cells with abnormal MEs. MEs of CCs and primitive stem cells such as embryonic stem cells and progenitor stem cells (PSCs) are abnormal due to association with telomerase [6]. The association with telomerase locks MEs in an exceptionally stable and active state to block terminal differentiation (TD) of cells with abnormal MEs [7,8]. DIs are chemical capable of eliminating telomeres from abnormal MEs, and DHIs are inhibitors of the ternary MEs consisting of methionine adenosyltransferase -methyltransferase-S-adenosylhomocysteine hydrolase [9]. It turns out that chemo-surveillance is in fact a natural mechanism to ensure wound healing, because the induction of TD of PSCs is a critical mechanism for the perfection of wound healing [2-3]. The functionality of chemo-surveillance becomes an important factor to dictate the success of wound healing [10,11]. If the functionality of chemo-surveillance is intact as healthy people, perfect wound healing can always be expected. On the contrary, if the functionality of chemo-surveillance has been damaged due to pathological conditions displaying cachexia symptoms, then the TD of PSCs will be impaired, thus allowing PSCs to evolve into CSCs, which requires a single hit to silence TET-1 enzyme. TET-1 enzyme is characteristically silenced in CCs [12-16]. The silencing of TET-1 enzyme is an easy task for PSCs to accomplish which are equipped with abnormally active MEs. The progression of CSCs to faster growing CCs can be achieved by activation of oncogenes through chromosomal translocations, or by inactivation of suppressor genes through chromosomal deletions or gene silences. It is clear that carcinogenesis involves complicated processes to display cachexia symptoms to cause the collapse of the functionality of chemo-surveillance that allows the evolution of CSCs from PSCs, and then the progression of CSCs to faster growing CCs by the activation of oncogenes and/or inactivation of suppressor genes. A perfect cancer drug must be able to resolve all these important issues of cancer. Wound healing metabolites that destabilize abnormal MEs to induce TD of PSCs and CSCs and CCs are apparently the best candidates to fulfill such requirements. Correction of gene abnormalities is evidently the most fascinating and attractive field of cancer research. Correction of gene abnormalities is, however, not easy. Even if a gene abnormality can be miraculously solved, there may soon pop up other gene abnormalities. It becomes endless struggles to solve difficult gene abnormalities. Induction of TD of CCs or killing of CCs are easy alternates to correction of gene abnormalities. Killing of CCs is out of question as above described. Induction of TD of CCs offers a promising prospective. After all, oncogenes and suppressor genes are cell cycle regulatory genes. They have important roles to play when cells are in cell cycle replicating. But if the replicating cells exit cell cycle to undergo TD, they have no roles to play. So a stroke to destabilize abnormal MEs to induce TD of CSCs and CCs can also put to rest problems arising from abnormal genes. No wonder it is the nature's choice to win the war on cancer [17,18]. Synthetic cell differentiation agent (CDA) formulations made up by DIs and DHIs, namely wound healing metabolites, plus phenyacetylglutamine as an anti-cachexia chemical are the right drugs to put away cancer permanently to save the lives of cancer patients [19]. There remains a big problem. Such therapy cannot make tumor to disappear. We have to establish a different criterion for the evaluation of therapeutic efficacy.

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