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Matching substrate provision and use to power cut oncogenesis

Abstract

Oncogenesis is driven by genetic and epigenetic alterations and occurs via gene expression dysregulation. Genotoxic agents, chromosome related abnormalities, pattern of methylation, diet and other environmental and nutritional factors contribute to carcinogenesis. This article innovatively describes how asynchrony between cell substrate availability and oxidation triggers known and unknown oncogenic mechanisms that may ultimately cause cancer.

Keywords: cell, oncogenesis, synchrony, substrate availability, oxidation

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Innovation and discussion

This perspective article establishes a relationship between the synchrony of cell substrate availability and use with development of cancer. Cancer is globally defined as augmented irregularities in cell physiology involving divisions and resulting abnormal genetics, epigenetics, genomics, proteomics and metabolomics (Figures 1).[1–3] The opportunity exists that establishing a harmony in substrate supply and the extent of substrate oxidation leads cells to be more biologically equipped and develop resistance against oncogenes.

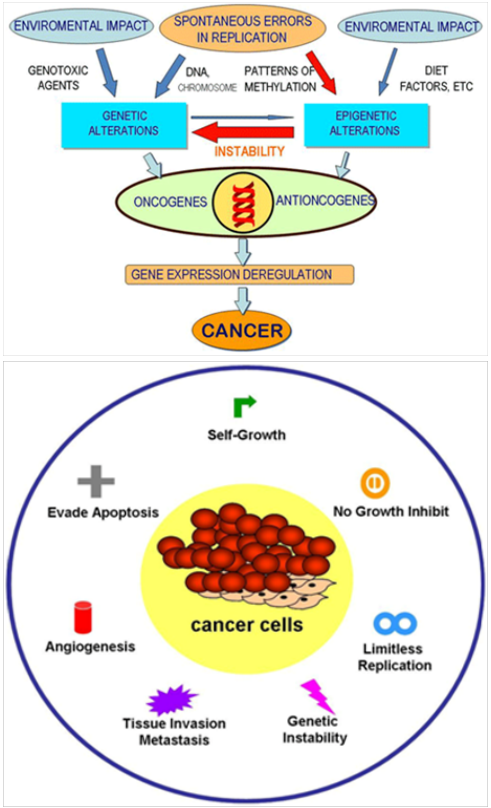

Figure 1 The collective contributions of genetic alterations and epigenetics on expression of genomic, proteomic and metabolomic dysregulations and oncogenesis leading to cancer.

Cancer is basically an abnormality that relates to regulation of tissue growth. For a healthy cell to transform into an unhealthy cancer cell, genes regulating cell growth and differentiation ought to be somehow changed. Genetic and epigenetic alterations may occur in varying levels and forms including gained, altered or lost chromosomes, and differing mutations influencing DNA nucleotides and silencing or over activating micro RNAs that oversee multitudes of genes expressions.

Oncogenes are either normal genes expressed at incongruously elevated levels, or somehow altered genes with novel characteristics. Classically, alterations in at least several genes must occur to transform a healthy cell into a cancer cell. The lower the chance of such alterations, the lower the risk of Oncogenesis. Timely nutrition and adequately coordinated and extensive physical activity must be pursued towards lowering the above risk in cancer development. This can be exercised through effective substrate oxidation and efficient waste management in different cells.

Looking at cancer in complicated ways would not allow its simplicity of action and expression to be evident. Moreover, focusing on what can really be accomplished successfully in a life time to prevent or at the very least slow down the Oncogenesis, is a key to smashing cancer. Apart from keeping the cells safe and away from devastating oncogenes such as viruses, bacteria, waves, and other environmental mutagens and carcinogens; nutritional manipulation and exercise are the most feasible pragmatic means to help prevent and manage cancer.

Based on recent discoveries, manipulating timing of nutrient supply to the body in ruminant animal models enables altering nutrient use efficiency and partitioning among oxidation, deposition, secretion, and excretion.[4–8] These findings imply that optimal synchronies in nutrient availability and use (e.g., oxidation, deposition) by splanchnic and peripheral cells may be achieved through shifting timing of nutrient intake and processing by different cells. As such, creating optimal synchronies between substrate delivery and utilization of mostly oxidation through timely nutrient intake and timely, but certainly, regular physical activity should keep Oncogenesis from development.

The main philosophy is that since food is consumed regularly and thus nutrients are supplied to the cells continuously and recurrently, nutrient oxidation and utilization must follow similar patterns. The

timely and corresponding nutrient oxidation and turnover can only be stimulated through adequate (and likely more than adequate) energy expenditure and substrate oxidation. Fuel use and oxidation is best stimulated by brain exercise and intense physical activity. Such a precise synchrony in nutrient delivery and oxidation rate must be realized on a circadian basis to permit daily high cell efficiency in crucial tissues such as brain, heart, liver, kidney and peripheral muscles. Collectively, the synchrony will favourably affect the aging process towards higher quality and sustainable lifestyle.

To elaborate on, due to individual and social differences in lifestyle including food regimens, exercise intensity, workload, sleep quality, exposure to stressors, socioeconomic status, educational perception, and the quality of the surrounding nature and environment, currently no ultimate quantitative recommendation on cancer prevention may be prescribed for a global use. However, a definitive feasible guideline is to match the extent of nutrient delivery with the level of nutrient oxidation and use by differential cells. Such a harmony must be accomplished at the very least on a circadian basis and not longer.

The synchronized daily management of cell nutrient availability and oxidation is a novel postmodern bioengineering of the overly hectic man exposed to a variety of carcinogens.

Implication

In a global pragmatic word, a simple but significant cause of genomic dysregulation is the impaired synchrony of substrate availability and oxidation in human cells. Among the pragmatic strategies preventing such an asynchrony or mismatch is timely and regular nutrition complemented by timely and regular but certainly challenging and demanding physical activity. These could ensure maintaining the regularity in cell physiology through avoiding substrate overload for considerable times over the lifespan.

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Conflicts of interest

The authors declare there is no conflict of interests.

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