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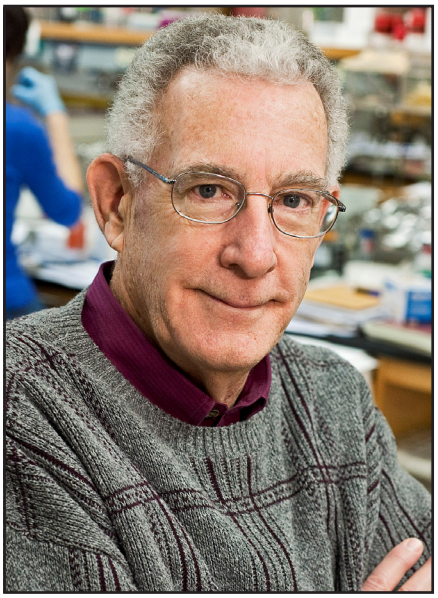
Cancer as a Metabolic Disease

excerpted from the book, "Cancer as a Metabolic Disease"
by Thomas N. Seyfried, Ph.D.

A major impediment in the effort to defeat cancer has been due, in large part, to the confusion surrounding the origin of the disease. "Make no mistake about it, the origin of cancer is far from settled." Contraindications and paradoxes continue to plague the field. Much of the confusion surrounding the origin of cancer arises from the absence of a unifying theory that can integrate the diverse observations on the nature of the disease. Without a clear idea on cancer origins, it becomes difficult to formulate a clear strategy for effective management and prevention. The failure to clearly define the origin of cancer is responsible in large part for the failure to significantly reduce the death rate from the disease.

Currently, most researchers consider cancer as a type of genetic disease where damage to a cell's DNA underlies the transformation of a normal cell into a potentially lethal cancer cell. The finding of hundreds and thousands of gene changes in different cancers has led to the idea that cancer is not a single disease, but is a collection of many different diseases. Consideration of cancer as a "disease complex" rather than as a single disease has contributed to the notion that management of various forms of the disease will require individual or "personalized" drug therapies.

This therapeutic strategy would certainly be logical if, in fact, most cancers were of genetic origin. What if most cancers are not of genetic origin? What if most of the gene changes identified in tumor tissue arise as a secondary downstream epiphenomena of tumor progression. What if cancer were a disease of respiratory insufficiency?



Although almost 700 targeted therapies have been developed from the cancer genome projects, no patients with solid tumors have been cured from this strategy.

The somatic mutation theory, which has guided cancer research and drug development for over half a century, is now under attack. Carlos Sonnenschein and Anna Soto along with others have identified major inconsistencies in the evidence supporting the genetic origin of cancer. Despite these concerns, the cancer field slogs forward with massive genome-based projects to identify all gene defects that occur in various tumor types.

In my opinion, it is wishful thinking that the vast information generated from the cancer genome atlas will someday serve as a foundation for the development of new and more effective cancer therapies despite recent arguments to the contrary. While gene-based targeted therapies could be effective against those few cancers that are inherited and where all the cells within the tumor have a common genetic defect, most cancers are not inherited through the germ line and few cancer cells have gene defects that are expressed in all cells of the tumor. Although almost 700 targeted therapies have been developed from the cancer genome projects, no patients with solid tumors have been cured from this strategy. How many times must we beat a dead horse before we realize that it will not get up and walk?

Most mutations found in tumors arise sporadically, as do most cancers. The types of mutations found in one tumor cell will differ from those found in another tumor cell within the same tumor. Genetic heterogeneity and randomness is the norm rather than the exception for

Yoga Research Society
341 Fitzwater Street
Philadelphia, PA 19147

phone: 215-592-9642
email: YRS@YogaResearchSociety.com
website: www.YogaResearchSociety.com

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mutations found in most sporadic cancers. We have recently shown how the majority of cancer gene defects could arise as downstream epiphenomena of tumor progression rather than as cancer causes. In light of these findings, it is not likely that gene-based targeting strategies will be useful for managing most advanced cancers.

Emerging evidence suggests that cancer is primarily a metabolic disease rather than a genetic disease. I will present evidence showing how cancer is a disease of defective cellular energy metabolism and that most of the genomic defects found in cancer cells arise as secondary downstream effects of defective energy metabolism. Most genetic defects found in tumors are "red herrings" that have diverted attention away from mitochondrial respiratory insufficiency, the central feature of the disease.

Regardless of cell type or tissue origin, the vast majority of cancer cells share a singular problem involving abnormal energy metabolism. While many in the cancer field consider gene defects as being responsible for the metabolic abnormalities in cancer cells, I do not share this view. I predict that targeting the defective energy metabolism of tumors will eventually become the most cost-effective, nontoxic approach to cancer prevention and management.

Dr. Thomas Seyfried will present "Cancer as a Metabolic Disease" at the 2016 YRS Conference.

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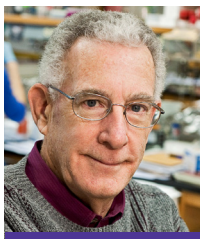

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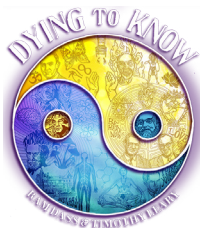

"Dying to Know"
narrated by
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