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Sarah S. Knox , Ph.D.

Executive Editor *of*

Journal of Integrative Oncology

Biography

Dr. Knox is a Professor at the University of West Virginia School of Public Health and the Mary Babb Randolph Cancer Center. She received her PhD and MS degrees from the University of Stockholm in Sweden and started her career as a Research Associate and then Associate Professor at the University of Stockholm where she was also Principal Investigator of her own research group at the Karolinska Institute of Environmental Medicine. Her research at that time focused on Swedish twins. After returning to the United States, she spent many years at the National Institutes of Health, first in an extramural and then in intramural capacity. She has been a scientific consultant to the World Health Organization and NATO, chaired national and international conferences and symposia and published and lectured extensively. Her current research interests focus on systems biology and biophysical signaling as they relate gene x environment interactions in cancer.

Research Interests

Systems Biology, Biophysics and Cancer

Recent Publications

1) **Knox SS**. Tumor Biology and Biophysics: A Systems Approach. *The International Journal of Molecular Medicine.* 2014;35 (Supple 1):S15.

2) **Knox SS,** Basu S, Remick S. A systems approach to cancer health disparities in Appalachia. *Austin J of Public Health*, 2014, 1(1):10.

3) **Knox SS**, Funk R. Oncology and Biophysics: The need for integration. *J of Clinical and Experimental Oncology*. 2014, S1, doi: 10.4172/2324-9110.S1-001.

4) **Knox SS**, Ochs MF. Implications of systemic dysfunction for the etiology of malignancy. *Gene Regulation and Systems Biology* 2013;7:11-22.

5) **Knox SS**. From "omics" to complex disease: a systems biology approach to gene-environment interactions in cancer. *Cancer Cell Int.* 2010; 10:11.

Systems Biology & Cancer

The mainstream view of cancer as a mutated cell that has become dysfunctional and multiplies out of control, is much less consistent with extant data than the context dependent model that focuses on interactions between the cell and its surrounding microenvironment as the initiator and driver of malignancy. The genome wide epigenetic changes that precede cancer and predict risk for cancer, strongly suggest that multiple systems are affected by gene expression changes before tumors ever manifest. This provides a partial explanation for cancer's ability to adapt to targeted therapies as well as an explanation for the failure of multiple DNA repair mechanisms and other defenses(e.g. apoptosis, immune defenses) to eliminate mutated cells. Dynamical systems theory can be utilized to explain the conundrum of tumors' ability to adapt to medications, and also as a model for the body's dynamic responses from a healthy attractor that attacks and eliminates abnormal cells, to a carcinogenic attractor which adapts to maintain malignancy.

A mechanism that is less well integrated into oncology research is biophysical signaling which plays a central role in cancer through influences on cell proliferation, cell cycle progression, apoptosis, cell migration and orientation, as well as cell differentiation. Serious progress in therapeutics will require integration of both biophysical and biochemical signaling in a systems biology approach to treatment.

Knox and Funk 2014

Related journals

- Chemotherapy: Open Access
- Journal of Leukemia

Related Conference

- Global Cancer Conference
- 19th World Congress on Advances in Oncology

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