

10th International Conference on
Genomics and Molecular Biology

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6th International Conference on
Integrative Biology

May 21-23, 2018 Barcelona, Spain

**Sarah S Knox***West Virginia University School of Public Health, USA***Biophysical signaling, systems biology and carcinogenesis**

Current clinical research in oncology has transitioned from a focus on generally cytotoxic chemotherapies to targeted, small molecule therapeutics. However, the broad array of gene repair and immune defense mechanisms in the body's arsenal against cancer indicate that cancer is not simply the result of a single renegade cell randomly mutating out of control. The fact that genome-wide epigenetic changes precede cancer, suggests that tumor etiology and progression involve multiple dynamical systems. One of the biological systems that have received the least attention in cancer research is that of the endogenous bioelectric signals stemming from ion channels in cell and mitochondrial membranes. These voltage potentials have been shown to play an important role in regulating cell differentiation, proliferation, migration, orientation, apoptosis and gene expression. In fact, one of the first steps in the epithelial-mesenchymal transition in tumor formation is cellular depolarization. Not only does hyperpolarization of oncogenes prevent tumor development, it has been demonstrated that bioelectric signals interact with biochemical signaling, and that depolarized V_{mem} is an epigenetic initiator of metastatic behavior even in the absence of a centralized tumor. The theme of this talk is that for cancer to occur, multiple systems must be dysfunctional and that biophysical signaling fills an important knowledge gap in current thinking in tumor biology.

Biography

Sarah S Knox completed her PhD at Stockholm University in Sweden and began her career at the Karolinska Institute. After returning to the US, she worked at the National Institutes of Health for 17 years. She is currently a full Professor at West Virginia University. Her publications, honors and awards can be found on Research Gate.

sknox@hsc.wvu.edu**Notes:**