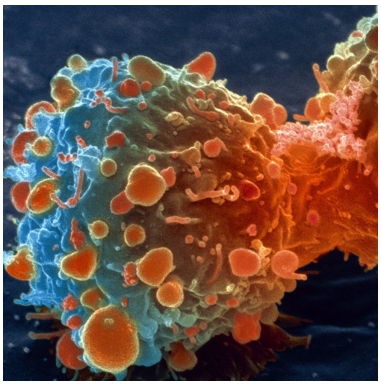
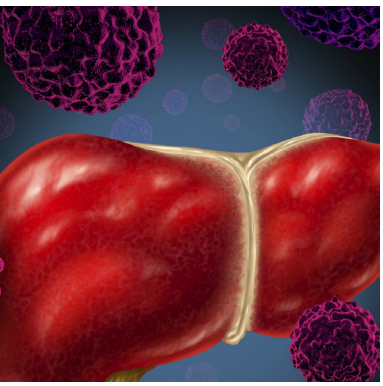
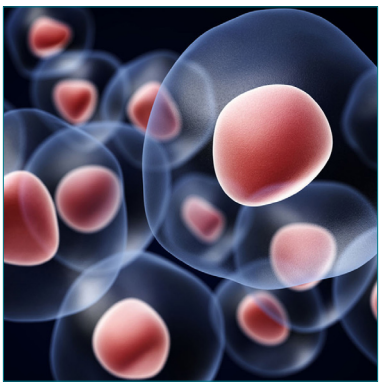
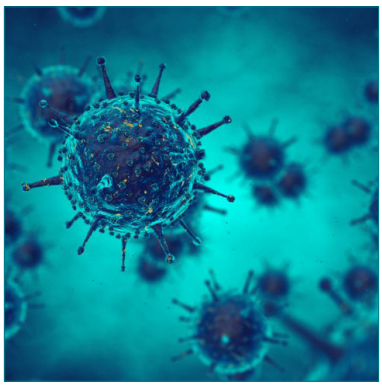
Keynote Forum
February 25-26, 2019

Cancer 2019



13th World Cancer Congress

February 25-26, 2019 | Paris, France

Stephen J Beebe

Old Dominion University, USA

Translational research with Nanosecond Pulse Stimulation for Immuno-Oncology applications

Nano-Pulse Stimulation (NPS) is a technology based on pulsed power physics, used for decades in high-powered physics and military applications. Electrical energy is stored and released in nanosecond bursts, producing instantaneous high power and low, non-thermal energy. Since biological cells have not experienced impacts like this in evolutionary history, they can exhibit unique intracellular responses that are noteworthy and remarkable. Under high NPS conditions tumor cells undergo programmed cell death (PCD) and innate and adaptive immune mechanisms are activated. Under low NPS levels cells can be stimulated and activated. The transition of this technology from physics scenarios to biological and medical landscapes uniquely combines expertise from engineers, physicists, biologists and physicians.

NPS strategy for cancer treatment uses 60-100 ns pulse durations and electric field strengths up to 50 kV/cm. When orthotopic mouse mammary and rat hepatocellular carcinoma tumors are eliminated by NPS, animals are protected by an immune-mediated, vaccine-like effect against exposure to the same cancer. Immune responses are dynamic on several therapeutic fronts. NPS directly eliminates primary tumors by inducing regulated form(s) of immunogenic cell death. This is accompanied by specific activation of subsets of CD8+ natural killer cells and NKT-cells expressing the NKG2D and CD161 activation receptors. In addition, dendritic cells (DCs), which are activated by dead and dying cancer cells, induce cytotoxic T-cells expressing adaptive memory phenotypes. Importantly, NPS eliminates immunosuppressive cells in the tumor microenvironment and blood. In the mouse model, an abscopal effect occurs including reduced spontaneous distant metastases and eradication of second untreated lesions.

Non-lethal NPS can activate DCs. NPS attenuates respiration in dendritic cells (DCs) and other cells by affecting complexes I and IV in the electron transport chain (ETC) increasing levels of superoxide anions in mitochondria, which presumably activate DCs as indicated by expression of activation markers and cytokine secretion. Higher NPS induces opening of the permeability transition pore and induces PCD. How these and other intracellular NPS-induced mechanisms lead to ablation-induced immune responses are under investigation.

Speaker Biography

Stephen J Beebe is a Research Professor in the Frank Reidy Research Center for Bioelectrics at Old Dominion University (ODU). He was a Fulbright and Marshall Scholar in Oslo, Norway. He is the author of 125 peer reviewed manuscripts and books chapters. He was awarded two NIH grants analyzing structure and function of Protein Kinase A and cAMP signal transduction. He now investigates mechanisms of NanoPulse Stimulation (NPS) in cancer and biology. He has trained over 30 graduate students and post-doctoral fellows, is a member of Editorial Boards for four journals and is the Chair of the ODU Institutional Animal Care and Use Committee (IACUC).

e: SBeebe@odu.edu

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Alvaro Macieira-Coelho

French National Institute of Health, France

The decline of the prevalence of Cancers during organism senescence

In general the scientific literature reports that aging favors the development of cancers. Each type of cancer however, initiates and evolves differently and their natural history can start way back at earlier ages before their clinical manifestations. The incidence of cancers is spread through the human life span, it is the result of pre- and post-natal aggressions, individual susceptibility, and developmental changes that evolve continuously from the beginning to the end. Finally during human senescence the incidence declines for all cancers. Frequently the progression of cancers is also slower in the old. There are several possible explanations for this decline at the tissue, cellular, and molecular levels. It is time to ask why some tumors are characteristic of the young, others of maturity, others of the time of the decline of the reproductive period, and finally why the incidence of cancers declines late during senescence of the human organism. These questions should be answered before the origin of cancers can be understood.

Speaker Biography

Alvaro Macieira-Coelho is a Research Director at the French National Institute of Health. He received an MD from the University of Lisbon, Portugal, and a PhD from the University of Uppsala Sweden. He made an internship at the University Hospital in Lisbon and was a research associate at the Wistar Institute in Philadelphia (USA) and at the Department of Cell Biology of the University of Uppsala (Sweden). He became Head of the Department of Cell Pathology at the Cancer Institute in Villejuif (France) and was a visiting Professor at the University of Linkoping (Sweden). He published 150 papers in professional Journals and 9 books on cancer and aging. He received the following awards: Fritz Verzar Prize (University of Vienna, Austria), "Seeds of Science" Career Prize (Lisbon, Portugal), Dr. Honoris Causa (University of Linkoping, Sweden), Johananof International Visiting Professor (Institute Mario Negri, Milano, Italy).

e: macieiracoelho@gmail.com

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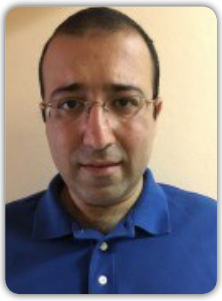

Nurettin İlter Sever

Pamukkale University, Turkey

The mysteries of S6K2 may shed light to Breast Cancer Therapy path

The divergence between S6 Kinase 2 (S6K2) and its homologue S6 Kinase 1 (S6K1) has displayed that the exclusive functions of S6K2 are very important mediators of tumor growth. Recent studies suggest that S6K2 complexes with B-Raf and PKCε to exert cancer cell survival. Also, indirect roles of S6K2 which involve interaction with Akt and PDCD4 to propagate cancer cell survival makes it an important therapeutic target. Also, centrosomal localization of a pool of S6K2 potentiates a proliferative role. Amplification and overexpression of *RPS6KB2* gene locus, which encodes S6K2 protein, is observed in breast cancer and is correlated with poor prognosis. Also, S6K2 expression is correlated with 4EBP1 and E2F1 expression in breast cancer. Also, breast cancer tissues display nuclear over-accumulation of S6K2 when compared to its normal counterparts.

Currently, the mechanisms which regulate the cellular levels of S6K2 are unknown. Also, there still remains new substrates of S6K2 to be unraveled. As the mysteries of S6K2 is solved, new stones are paved in the breast cancer therapy path.

Speaker Biography

Nurettin İlter Sever has completed his PhD in 2013 from The Ohio State University, USA. He is currently an assistant professor at Pamukkale University, Denizli, Turkey. He is currently establishing his laboratory and is a member of EACR.

e: nsever@pau.edu.tr

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