Onsite **Program**

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ISBN: 978-1-988006-00-0

SESSION TIME: **17:00 – 18:15**

SESSION ROOM: **716B**

SESSION TRACK: **TRACK 19: BIOPHYSICS AND MODELLING**

SESSION NAME: **SP159 – TRANSPORT AND PHYSIOLOGICAL MODELLING**

SESSION CHAIR(S): **CHAI HONG YEONG, MALAYSIA**

17:00 SP159.1 - **KEYNOTE:** Dwarfing Big Data for Oncology Applications: Necessity and Possibilities
Issam El Naqa, Canada

17:30 SP159.2 - Improved temperature monitoring and treatment planning for loco-regional hyperthermia treatments of Non-Muscle Invasive Bladder Cancer (NMIBC)
Gerben Schooneveldt, Netherlands

17:45 SP159.3 - A Full 3D CFD Model Coupled with an Outflow Lumped Boundary and Inflow Total Pressure Formulation to Estimate Human Cardiac Perfusion
Iyad Fayssal, Lebanon

18:00 SP159.4 - Simulation Model of Image-Guided Percutaneous Thermal Ablation in the Assessment of Optimal Approach for Complete Tumour Ablation
Chai Hong Yeong, Malaysia

SESSION TIME: **17:00 – 18:15**

SESSION ROOM: **713B**

SESSION TRACK: **PRESIDENT'S CALL**

SESSION NAME: **SP160 – NEUROENGINEERING, NEURAL SYSTEMS / BIOPHYSICS AND MODELLING**

SESSION CHAIR(S): **VENKATESHWARLA RAJU, INDIA**
TEODORO CORDOVA - FRAGA, MEXICO

17:00 SP160.1 - From 'Fracking' and 'Macrovoids' to the Onset of Cancer Metastasis: A Mechano-Metabolomics Model of a Plausible Fluid-Solid Network Instability in Tumors
Sai Prakash, United States

17:15 SP160.2 - Surface electromyography in quantifying Parkinson's disease and its treatment with deep brain stimulation
Pasi Karjalainen, Finlandia

17:30 SP160.3 - A Decade of Experience with Intraoperative Microelectrode Recording in Determining the Subthalamic Nuclie (STN) Deep Brain Stimulation? Lead Positions in 260 Parkinson Diseased Conditions in South India? A Retrospective Study
Venkateshwarla Raju, India

17:45 SP160.4 - Vortex of the Magnetic Field on the Growth Rate of Escherichia Coli
Teodoro Cordova - Fraga, Mexico

18:00 SP160.5 - Electro Magnetic Therapy and Laser in the Chronic Pain Of The Woman
Manuel Zuniga, Ecuador

SP160 - Neuroengineering, Neural Systems / Biophysics And Modelling

PRESIDENTS CALL

SP160.1 - From "Fracking" and "Macrovoids" to the Onset of Cancer Metastasis: A Mechano-Metabolomics Model of a Plausible Fluid-Solid Network Instability in Tumors

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Based on analogy and inductive reasoning, we postulate that a malignant tumor (carcinoma) grows in size until the "de-percolation" threshold of its encapsulating basement membrane (BM) which undergoes proteolysis during oncogenesis. Typically, BMs are composed of tri-continuous hydrogel networks of collagen-IV, laminin, and interstitial fluid, with connector proteins such as nidogens, and perlecans. The aforementioned threshold is determined by the mechanochemical state of the tumor-membrane complex vis-à-vis its microenvironment and, herein, conjectured to synchronize with the tensile yielding/rupture of the BM. Thereupon, peripheral cells of the tumor undergo epithelial-to-mesenchymal transitions (EMT), becoming motile, and migratory.

We test this postulate by formulating a mathematical model based on continuum fluid-solid mechanics, diffusion, and cell bioenergetics theories. This approach may be characterized as "mechano-metabolomics" modeling at the continuum scale and complements genomic, and proteomic approaches. In this model, a prototypical, viscous tumor spheroid grows radially, consuming metabolic nutrients (viz., glucose, oxygen, and lactate) while being constrained by an elastic BM *ca.* 0.5-2 microns-thick, and a network of linkages of cell adhesion molecules (CAMs), chiefly cadherins and integrins. A fundamental schema "induced" from geology and polymer physics is the fluid-solid mechanical network instability observed in rock formations during hydraulic fracturing (fracking) and during formation of synthetic asymmetric membranes (vis-à-vis large voids), respectively. Polymer physics also furnishes scaling concepts to develop equations of state for cells, and concepts describing cell and tissue rheology via gel poroelasticity theory. Besides, critical perspectives from other recent developments, viz., homeorhetic tumor pressures, mechanotransduction, and symbiotic glucose-lactate metabolism observed in cancer cells, are incorporated, to base the framework on well-grounded physicochemical principles in abiological and/or microbial systems. The nonlinear model is computationally analyzed via Comsol Multiphysics®.

The theoretical simulations ("*in silico* experiments") lucidly support the *a priori* conjecture and are consistent with biological observations of oncogenesis *in vivo*, and physicochemical measurements *in vitro*. Computed stress-strain fields in the tumor microenvironment suggest that proteolyzed BMs, quantified by decreased elastic moduli, participate in aberrant tumor growth dynamics and likely undergo tensile rupture and stress localization-induced cellular detachments, initiating EMTs, and metastasis. Subsequent crack-tip stresses may also shift strains on CAMs from compressive to tensile, suggesting mechanotransduced conformational switches, such as from non-invasive, adherent E-cadherins to invasive N-cadherin phenotypes.

The model also provides a rationale to detect metastatic potential of tumors via a convenient diagnostic imaging tool such as positron emission tomography (PET). PET locates abnormally-high glucose uptake rates in cells and tissues, which are related to the mechanical field evolution of the tumor microenvironment, and thereby, the likelihood of BM rupture. Complementarily, newer techniques that characterize tissue elasticity such as elastography may also be applied in concert with the developed framework.

A major conclusion of this research is that a hallmark of cancer may be a measurable, microscale "physical event" in contrast to nanoscale genomic or proteomic events. Besides proteolysis as a "proximal cause" to rupture, other cancer phenotypes such as higher proliferation rates, softer cells, fractality, etc., can also be incorporated into the model. Experimentation is required to verify the postulate.

SP160.2 - Surface electromyography in quantifying Parkinson's disease and its treatment with deep brain stimulation

Author(s): Saara M. Rissanen[1], Zhe Li[2], Markku Kankaanpää[3], Jian Wang[4], Olavi Airaksinen[2], Pasi A. Karjalainen[1]
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Parkinson's disease (PD) is a progressive neurodegenerative disorder that affects millions of people worldwide. The disease cannot be cured but the motor symptoms can be relieved with medication or with deep brain stimulation (DBS). However, there is a lack of objective and quantitative methods for monitoring PD and the efficacy of its treatment. Finding these objective methods is a global challenge.

Surface electromyography (EMG) and a principal component (PC) -based monitoring method was used here for quantifying PD-characteristic features in the EMG signals. The aim was to find the most effective combination of EMG signal features for characterizing PD and its treatment with DBS. It was quantified, if the EMG signals of PD patients change into more similar with the signals of healthy subjects with DBS.

Two groups of PD patients (9 patients with DBS ON and OFF, and 22 patients with medication OFF) and one group of healthy subjects (13 age-matched subjects) were measured during isometric contraction of biceps brachii (BB) muscles and during elbow flexion-extension movements. Surface EMGs were recorded from BB muscles. Several parameters were calculated from the EMG signals. The following parameters worked best in discriminating between healthy controls and PD patients: recurrence rate, correlation dimension, sample kurtosis and crossing rate variable of EMG during isometric contraction, and EMG burst frequency, sample kurtosis and recurrence rate of EMG during extension movements. These variables were chosen to form feature vectors for each subject in the PC-based monitoring method. The analysis revealed clear differences in the EMG features between healthy controls and PD patients (see Fig. 1), and also between DBS OFF and ON. In eight out of nine patients, the surface EMG features changed into more similar with the features of healthy controls when the stimulator was switched on.

The results showed that surface EMG is capable of characterizing patients with PD and effects of DBS treatment. Surface EMG is therefore a potential method for monitoring PD and its treatment efficacy objectively and quantitatively.