

Monday 10th September SYSMED 2012 Conference (inclusive of Sponsor Exhibition in Carton Suite I) Carton Suite, Conference Centre

- 8.30 9.30 Registration
- **9.30 10.00** Conference Opening and Welcome Desmond Fitzgerald, University College Dublin, Ireland Christina Kyriakopoulou, Health Directorate, DG Research & Innovation, European Commission, Belgium

10.00 - 13.10 Session I: Systems Medicine - Hype or Hope?

- Chair: Desmond Fitzgerald, University College Dublin
- 10.00 10.40 Systems Medicine: How to Capitalise on Molecular Medicine Hans Westerhoff, Manchester Centre for Integrative Systems Biology (MCISB), University of Manchester, UK/VU University Amsterdam, Netherlands/IT Future of Medicine
- 10.40 11.10 Coffee break
- 11.10 11.50 Systems Pathology: How much more can tissue tell us? David Harrison, St Andrew's University, UK
- 11.50 12.30 The way to Systems Medicine Approaches in neuroblastoma Angelika Eggert, Department of Pediatric Hematology/Oncology, Pulmonology and Cardiology, University Hospital of Essen, Germany
- 12.30 13.10 Systems Biology and Translational Medicine- Maximising the Synergy Seamas Donnelly, Education and Research Centre, St. Vincent's University Hospital and School of Medicine, University College Dublin, Ireland
- 13:10 14:00 Lunch in The Linden Tree Restaurant
- 14.00 16.00 Session II: Frontiers in Medicine

Chair: Pierre De Meyts, De Meyts R&D Consulting SPRLU, Belgium

- 14:00 14:40 Interactome Networks and Human Disease Marc Vidal, Centre for Cancer Systems Biology, Department of Genetics, Dana-Farber Cancer Institute/Harvard Medical School, Boston, USA
- 14.40 15.20 Cancer Response to Targeted Agents: Dynamics and Variability from Single Cell Data Vito Quaranta, Integrative Cancer Biology Center, Vanderbilt University Medical School, Nashville, USA
- 15.20 15.40A Brief View of Systems Medicine and GeneticsJie (Bangzhe) Zeng, Institute of Systems Biological Engineering, China
- 15.40 16.00 Pathway-GPS and SIGORA: Identifying relevant pathways based on the overrepresentation of their gene-pair signatures David Lynn, Teagasc, Ireland
- 16.00 16.30 Coffee break

16.30 - 20.00 Panel Discussion, Exhibition and Networking Session

16:30 - 16:50 Introduction to panel discussion Adriano Henney, German Virtual Liver Network, University of Heidelberg, Germany (Chair)



count data, collected every 6 minutes, are initially fit with a novel Quiescence-Growth mathematical model, based on three parameters: division, death and quiescence rates. This model is then substantiated by extracting these rates from experimental observations of hundreds of single cells, fitted with an Exponentially Modified Gaussian model. In the final output graphs, Fractional Proliferation describes the underlying behavioral dynamics that result in proliferative changed by perturbations. Using this method, we discovered that the response of lung cancer cell lines to erlotinib, an epidermal growth factor receptor tyrosine kinase inhibitor, is a nonlinear process dominated by an increased rate of cell entry into quiescence. Even in highly sensitive, homogeneous "oncogeneaddicted" cell populations, we observed variability of cell-to-cell response. Up to 72 hours of treatment, quiescence prevailed, with only a modest increase in death rate. After 72 hours, the population of treated cells reaches a new steady state in the presence of erlotinib characterized by an overall rate of proliferation, which is a composite of death, guiescence and division rates. Similar results were obtained with oncogene-addicted melanoma and breast cancer cell lines treated with the respective targeted oncogene inhibitors. In contrast to our results, drug targeting of addicting oncogenes has thus far been thought to result in massive cell death. Instead, our findings indicate that it may cause response behaviors other than death, underscoring the realistic in vitro representation of cell proliferative response to perturbations provided by Fractional Proliferation, and providing means to optimize and improve discovery and deployment of targeted therapy in cancer.

A Brief View of Systems Medicine and Genetics

Jie (Bangzhe) Zeng, Xiao-Xue Zeng

Institute of Systems Biological Engineering, China

To investigate natural and artificial biosystems, systems biology which included systems genetics and systems medical study of diseases, systems biological engineering worked as synthetic biology and systems biotechnology, were established on the theoretical fundamentals of evolution, systems and structure theories. The science and engineering of biosystems are disciplines by integrative methodology of computational, experimental and engineering biology. Systems dynamics of cytogenesis and bio-molecular networks is explored as the mechanism for the evolution of genomic structures and cell lineages mapping during pattern formation of organisms. For drug discovery, micro-fluidic chips are useful for the functional analysis, identification of expression genes among difference cell-types and drug treatments, and also used for the designing of artificial cells such as neurons and neuronal communication networks etc.

Refs:

1. Zeng (B.) J., On the holographic model of human body, 1st National Conference of Comparative Studies Traditional Chinese Medicine and West Medicine, Medicine and Philosophy, April, 1992 (on the concept of systems medicine).

2. Zeng (B.) J., On the concept of system biological engineering, Communication on Transgenic Animals, No. 6, June, 1994.

3. Zeng (B.) J., Transgenic animal expression system – transgenic egg plan (goldegg plan),

Communication on Transgenic Animal, Vol.1, No.11, 1994 (on the concept of system genetics).

4. Zeng (B.) J., From positive to synthetic medical science, Communication on Transgenic Animals, No.11, 1995 (on systems medicine).

5. Zeng(B.)J., The structure theory of self-organization systems, Communication on Transgenic Animals, No.8-10, 1996. Etc.