Scientific Symposium (Mon, 26 Sep, 14:45−16:45)
High Throughput Technology Platforms for Biomarker Discovery − State of the Art

345 INVITED
Overview and Definition of Biomarkers (General Introduction)

Abstract not received

346 INVITED
Role of Gene Expression Profiling for Biomarker Discovery

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Gene expression profiling studies have made some important contributions to biomarker discovery, particularly in breast cancer, and in the process the limitations of the technology has also become clearer. Gene expression profiling using DNA microarrays is a powerful and robust technology to identify disease subsets that have large scale molecular differences. As a result of these studies it is now widely accepted that breast cancer is not a single disease with variable Estrogen Receptor (ER) expression and histology; but ER-positive and -negative cancers represent two fundamentally distinct diseases. Similar observations were made in other cancers including lymphomas, lung and prostate cancer although the clinical relevance of these new classification schemas is less well established for these cancers. The large mRNA expression footprint of proliferation and ER-signaling also proved useful to develop new multi-gene prognostic tests for ER-positive breast cancers that are now used in the clinic to aid adjuvant adjuvant treatment selection. However, it is also increasingly clear that gene expression profiling is not be able to identify small scale and variable mRNA expression differences that may be associated with many clinically relevant prediction problems. Robust prognostic signatures for triple-negative breast cancer or predictive-signatures with clinically relevant accuracy for any individual drug in any cancer remain elusive goals despite 15 years of research. An important lesson learned from these studies is that clinically important functions may be associated with limited and inconsistent mRNA expression footprints and substantially larger sample sizes will be required to solve these classification problems.

347 INVITED
Cancer Proteomics − From low Resolution to High Resolution to Study Lung Cancer Phenotype

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Proteome analysis aims to study the proteins on a systemic level in contrast to focusing the investigation on only one or a few proteins at a time. Proteomics is a collective term for techniques used for analysis of a proteome. In recent years, the development of both mass spectrometry (MS) based and antibody based proteomics methods has been tremendous. These advances in discovery proteomics have been propelled by improvements in instrumentation, sample pre-fractionation methods and improved bioinformatics pushing the field towards biological and clinical research. We have developed and used these methods especially to study clinical materials to understand how cancer cells responds to treatment and what, on the proteome level, defines a highly malignant cancer phenotype.
One of the major drawbacks in proteomics has been the lack of analytical depth, especially when studying complex proteomes such as the human proteome. This is manifested by lack of data on central regulators in cancer, such as transcription factors, their co-factors, tyrosine kinase receptors etc., in proteomics experiments. We have developed methods to increase proteome coverage in quantitative proteomics experiments on clinical material using high resolution fractionation (peptide isoelctric focusing) followed by mass spectrometry analysis. Here we present how this major leap in analytical depth in proteomics enables us to detect and study cancer pathways related to metastatic phenotype, cancer metabolism and therapy response to EGFR inhibition with focus on lung cancer.

348 INVITED
Full Genome Sequence Analysis

Abstract not received

349 INVITED
How Do We Study Network Pertubations in Clinical Specimens? How Do We Select "Drivers" of Malignancies?

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We need to develop an open innovation space where physicians, patients and scientists can together develop maps of cancer capable of driving better screening, diagnosis, treatment, and control of cancer. We will need to develop an infrastructure to manage this data and to provide an environment to build these maps of disease. This is not a problem that is solved by few but will involve large-scale involvement of the scientific and patient communities working together. Here are some of the issues to be discussed:

- Accessible but minimally usable clinical/genomic data- little care to annotate and curate data for other's use
- Mathematical models of disease are not built to be reproduced or versioned by others
- Data seen as supplemental materials after publications
- Assumption that those funded to generate data somehow own the data they generate
- Assumption that genetic alterations in human tumours can be owned
- Transient nature of sites where data models and tools for others are maintained
- Lack of standard forms for sharing data and future rights
- Most patients are not actively participating in donating samples and their outcome data
- Few cancer patients as activists demanding sharing data in the public forum
- Most clinical/genomic data generated by industry from trials is not shared
- Most academics feel they need to sequester data until their lab can complete publishing
- Rewards are for first/last authors who want to protect their unique contribution till after full article is published.

Scientific Symposium (Mon, 26 Sep, 14:45−16:45)
Drug Development in Paediatric Oncology

350 INVITED
4 Years Later − the Impact of the European Pediatric Medicine Regulation on Children and Adolescents in Europe

Abstract not received

351 INVITED
PARP Inhibition in Pediatric Malignancies

Abstract not received

352 INVITED
Targeting ALK in Paediatric Malignancies

Abstract not received

353 INVITED
Immunotherapy of Paediatric Malignancies

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Two major concepts have been recently identified in tumour immunity: the tumour immunoediting and the therapy-induced immunogenic cell death. Tumour immunoediting refers to 3 phases designed elimination, equilibrium, and escape. In the escape phase, tumour cells circumvent both innate and adaptative immune defences either by alteration occurring in edited tumour cells themselves, or by inhibition of the protective functions of the immune system, or by the generation of immunosuppressive cells. When cancer cells die through immunogenic cell death, they also alert the immune system, which mounts a therapeutic anti-cancer immune response and contributes to the control of residual tumour cells. Radiotherapy and some chemotherapy agents, in particular anthracyclines, can induce specific immune responses that result either in immunogenic cell death or in immunostimulatory side effects. Paediatric tumour immunology is of particular interest because (i) the environmental factors appears to play a minimal role in the genesis of cancer suggesting a more important role of host-related factors when compared with adult cancers, (ii) most of the tumours occur within the first years of life suggesting a special and