8th EACR-OECI Joint Course

Molecular Pathology Approach to Cancer

04 - 06 June 2018
Amsterdam, Netherlands

Scientific Programme Committee
Leonor David || Richard Marais || Jorge Reis-Filho
Giorgio Stanta || Marc van de Vijver

Programme Book
### Day 1 - Monday 04 June

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<td>NETWORKING RECEPTION</td>
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<td>Welcome drinks and a hot buffet dinner will be served for all participants and exhibitors to enjoy. The trade exhibition will be open at this time, and there is an opportunity to continue networking.</td>
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Day 2 - Tuesday 05 June

08.30 – 09.00  MORNING COFFEE Oosterhuiszaal and Rode Hoed foyer
Coffee available to purchase from the bar

09.00 – 09.20  Marc van de Vijver AMC & VU Medical Center, Netherlands
“The molecular pathology of breast cancer”
Q&A: 09.20 – 09.30

09.30 – 09.50  David Huntsman UBC / BC Cancer Agency, Canada
“The molecular pathology of ovarian cancer”
Q&A: 09.50 – 10.00

10.00 – 10.20  Britta Weigelt Memorial Sloan Kettering Cancer Center, USA
“The molecular pathology of endometrial cancer”
Q&A: 10.20 – 10.30

10.30 – 11.00  COFFEE BREAK Oosterhuiszaal and Rode Hoed foyer

11.00 – 11.20  Daniel Peeper NKI, Netherlands
“The molecular pathology of melanoma”
Q&A: 11.20 – 11.30

11.30 – 11.50  Andreas Jung LMU Munich, Germany
“The molecular pathology of colorectal cancer”
Q&A: 11.50 – 12.00

12.00 – 12.30  SATELLITE SYMPOSIUM - ELITE SPONSOR

12.30 – 13.00  LUNCH Oosterhuiszaal and Rode Hoed foyer

13.30 – 13.50  Brian Rubin Cleveland Clinic/Lerner Research Institute, USA
“The molecular pathology of GISTS”
Q&A: 13.50 – 14.00

14.00 – 14.20  Judith Bovee Leiden University Medical Center, Netherlands
“The molecular pathology of bone tumours”
Q&A: 14.20 – 14.30

14.30 – 14.50  Matt van der Rijn Stanford School of Medicine, USA
“The molecular pathology of soft tissue sarcomas”
Q&A: 14.50 – 15.00

15.00 – 15.30  COFFEE BREAK Oosterhuiszaal and Rode Hoed foyer

15.30 – 15.50  Marc Ladanyi Memorial Sloan Kettering Cancer Center, USA
“The molecular pathology of lung cancer”
Q&A: 15.50 – 16.00

16.00 – 16.20  Caroline Dive CRUK Manchester Institute, UK
“CTCs: a multi-use liquid biopsy”
Q&A: 16.20 – 16.30

16.30  FREE EVENING TO EXPLORE AMSTERDAM
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<td>Pieter Wesseling VU University Medical Center, Netherlands</td>
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<td><strong>“The molecular pathology of the central nervous system tumours”</strong></td>
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<td>Giorgio Stanta University of Trieste, Italy</td>
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<td><strong>“The evolution of pathology”</strong></td>
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<td>GENOMICS TESTING FOR CLINICAL PRACTICE</td>
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<td>Marc Ladanyi Memorial Sloan Kettering Cancer Center, USA</td>
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<td>David Huntsman UBC / BC Cancer Agency, Canada</td>
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Congratulations to the winners of the EACR-Worldwide Cancer Research and OECI Meeting Bursaries. Each winner received a full registration free of charge and funds of up to 500 Euros to assist with the cost of travel and accommodation.

**EACR-Worldwide Cancer Research winners**
- Muhammed Khaled Elfaituri, Libya
- Mariia Inomistova, Ukraine
- Anna Khokhlova, Russia
- Jure Krasić, Croatia
- Vikrant Palande, Israel
- Sotiria Stasinopoulou, Greece

**OECI winners**
- Tetiana Borikun, Ukraine
- Yasemin Cakir, Turkey
- Alessio Menga, Italy
- Hanno Roomere, Estonia
- Alexander Scherbakov, Russia

**Course evaluation, CME credits and Certificate of Attendance**

Once the meeting has concluded, an online survey will be sent requesting participants’ evaluation and feedback on the course. A Certificate of Attendance conveying CME Credits will be available to download and print on completion of the online Evaluation Survey.

The 8th EACR-OECI Joint Course: Molecular Pathology Approach to Cancer, Amsterdam, Netherlands, 04/06/2018-06/06/2018 has been accredited by the European Accreditation Council for Continuing Medical Education (EACCME®) with 10 European CME credits (ECMEC®s). Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity.
Molecular Pathology: why bother?

Jorge Reis-Filho  
Memorial Sloan Kettering Cancer Center, USA

Pathology, as a medical specialty, is changing profoundly. Although originally conceived as the science devoted to understanding the mechanisms of disease, in the 20th Century, Pathology came to become a medical specialty primarily devoted to the diagnosis of human illnesses on the basis of histologic analyses of human tissues. For many, the primary role of diagnostic surgical pathologists is to guide the surgeon’s hands; however, this is longer suffices for therapy decision-making. The advent of molecular tools for the characterization of human tumors and the emergence of the concept of precision medicine, information above and beyond that offered by histologic analyses of human cancers has become essential. Massively parallel sequencing has allowed for the characterization of the genomes of human cancers at base pair resolution, the identification of therapeutic targets and predictors of response to specific therapeutic agents, including immunotherapy approaches. Sequencing analysis currently provides information above and beyond tumor genotyping and the identification of copy number alterations; in fact, sequencing data can be used to define the mutational and rearrangement signatures of cancers, which provide important information about the biological processes that shaped the genomes of cancer cells and may inform therapy decision-making. Pathology, given its position at the interface between basic sciences and the delivery of patient care, is central to scientific endeavors devoted to the characterization of human cancers and the identification of diagnostic and predictive biomarkers. In this presentation, the impact of pathology in the design and interpretation of genomics studies and the role of the pathologist in precision medicine multidisciplinary teams will be discussed.
Massively-parallel sequencing

Serena Nik-Zainal
Sanger Institute, UK

The recent increase in the speed of sequencing offered by modern sequencing technologies permits an unprecedented degree of exploration of the human genome. No longer are we restricted to PCR-defined fragments of protein-coding exons, we can now investigate all the genetic material in human cells. I explain the principles underlying massively-parallel sequencing giving some insight into the advances as well as the difficulties posed by processing of the enormous datasets generated by modern sequencing experiments.

Cancer is the ultimate disorder of the genome, characterised by not just one or two mutations, but hundreds to thousands of acquired mutations that have been accrued through the development of a tumour. Utilising the extraordinary surge in scale as well as the digital nature of massively-parallel sequencing, I explain some of the recent highlights into tumour biology offered by these modern methods: cancer gene discovery, mutation signatures and cancer evolution.

Background reading:

Cancer genomics background

Massively-parallel sequencing and more recent technologies

Impact of NGS: reviews

Signatures of mutagenesis

Cancer evolution
Signalling and signal transduction for beginners

Britta Weigelt
Memorial Sloan Kettering Cancer Center, USA

Cancers are driven by genetic and/or epigenetic alterations, which often map to signaling pathways that control cell growth and division, cell death, differentiation and cell motility. The general principles of signal transduction, namely the transmission of signaling information from the receptor at the cell surface across the cytoplasm to the nucleus (gene transcription) or the cytoskeleton (cell motility), will be discussed. Furthermore, key signal transduction pathways that are altered in cancer cells, including the PI3K-AKT and MAPK pathways, and their activation through mutations affecting proto-oncogenes or tumor suppressors will be presented. Finally, the role of signaling, crosstalk between signal transduction pathways and feedback loops in the resistance to targeted therapeutics will be discussed.

Background reading:

Whole genome sequencing of metastatic cancer

Edwin Cuppen
Center for Personalized Cancer Treatment, Hartwig Medical Foundation and UMC Utrecht, Netherlands

Next-generation DNA sequencing has boosted the promises of personalising cancer treatment. It has now become possible to routinely sequence the complete genome of a tumor from a patient. While patient stratification based on a limited number of genetic measurements is steadily increasing in routine diagnostics, retrospective systematic analyses of genetic information and treatment outcome are still warranted to improve such stratifications, as significant numbers of selected patients for specific treatments are non-responsive, yet may experience severe treatment side effects. In addition, ineffective treatment contributes significantly to the increasing economical burden of novel cancer treatment drugs on health care costs.

As a national initiative, the Hartwig Medical Foundation (HMF) provides high-quality whole genome sequencing (WGS) for two large Dutch clinical studies: the Center for Personalized Cancer Treatment (CPCT-02) study and the DRUP (The Drug Rediscovery Protocol) trial, in which more than 45 hospitals participate. Sequencing results are used for building of a large database of genetic characteristics of metastatic cancers and patient data, including treatment and treatment outcome, and to generate a comprehensive patient report for supporting treatment choice.

Fresh-frozen tumor biopsies are collected and confirmed by central pathology to contain sufficient tumor cells (≥30%). At least 50 ng of DNA is required for WGS using an Illumina X10 setup in a dedicated ISO accredited laboratory. For all patients, tumor sample(s) as well as a blood sample are analyzed to identify all somatic aberrations. An in-house bioinformatic pipeline has been established and all scripts are publicly available at GitHub. Median turnaround time from biopsy to sequencing report is 25 days, and ~40 patients are processed every week.

Currently, more than 2,500 tumor biopsies have been successfully sequenced from patients with advanced metastatic disease who are either receiving (last line) regular therapy or for whom all regular options have been exhausted.

With an average sequencing depth of >100x, WGS enables sensitive identification of genome-wide somatic variants, indels, and copy number variants. Gene fusions and structural variations (SV) (e.g. BRCA inactivation by SV instead of a mutation) are detected, which cannot be identified reliably using gene panels or exome sequencing. In addition, tumor cell purity is calculated from the sequencing data, allowing accurate determination local ploidy for each mutation, loss-of-heterozygosity (LOH) and sub-clonal events, which is essential for proper functional interpretation. All data is deposited in the HMF database and investigators can request access to answer specific research questions.

The HMF patient report provides an overview of all aberrations in 124 cancer related genes. In addition, higher-level molecular features such as mutational load and
microsatellite (in)stability are provided. To support interpretation of the observed variants, the report provides evidence items from the Clinical Interpretation of Variants in Cancer (CIViC) knowledgebase and clinical trials for which the patients is potentially eligible.

Taken together, WGS provides a complete view of all aberrations of a tumor including the mutational load and microsatellite status. Moreover, information on LOH, tumor purity and variant ploidy, gene fusions and other SV types are provided, all using a single biopsy. Together, this data facilitates new biomarker discovery, and the best possible treatment decisions or clinical trial inclusion.

Background reading:

Biomarkers as readouts of gene expression regulation

Leonor David
University of Porto and IPATIMUP/I3S, Portugal

The first cancer biomarkers to be identified, and still widely used in the clinic, are mucins or mucin glycoproteins (e.g. MUC1-CA15.3; MUC16-CA125; SLea-CA19.9) that were discovered by immunizing mice with whole cell extracts from cancer cells, and later spotted by antibody recognition strategies. Most are cell surface biomarkers, not exclusive of cancer cells but overexpressed and modified by altered glycosylation in cancer. They are not, in most instances, the direct result of genetic alterations. In contrast, we are starting to understand how cell surface biomarkers develop under the regulation of gene expression, mostly under lineage dependent programs orchestrated by homeobox genes. We envision that getting more into this new information will help us not only to refine and improve biomarker specificity by understanding post-translational modifications, but also to tackle lineage dependence in tumour oncogenesis and to generate new therapeutic approaches that incorporate complex genetic backgrounds and lineage dependence in a single shot.

Background reading:

ctDNA to support clinical decisions for cancer patients

Caroline Dive
CRUK Manchester Institute, UK

Content not available at the time of printing.
The molecular pathology of breast cancer

Marc van de Vijver
AMC and VU Medical Center, Netherlands

Breast cancer is presently classified based on tumor diameter, histologic type and grade, lymph node status and estrogen receptor, progesterone receptor and HER2 status. This classification has important implications for the surgical, radiotherapy and systemic treatment of breast cancer patients.

Genomic characterization of breast cancer has provided a wealth of additional information on breast cancer biology, molecular pathology of breast cancer is becoming increasingly important and several molecular breast cancer tests have been implemented in clinical practice over the last years.

Based on histological features, breast cancer is categorized as invasive ductal carcinoma, comprising approximately 70% of all cases; invasive lobular carcinoma, comprising approximately 10% of all cases; and several special and rare types, together comprising 20% of all cases. This histologic classification can be supplemented with categories based on genetic alterations; and categories based on gene expression profiles. Whole genome sequence data will provide the next supplement to an even more refined classification of breast cancer.

The genetic alterations identified in breast cancer are amplification of between 10 and 20 oncogenes (or genomic regions with as yet not an identified “driving” oncogene) and mutations in oncogenes and tumor suppressor genes. Over 2,000 breast carcinomas have been subjected to whole genome sequence analysis or whole exome sequence analysis. From this work it has become clear that there are only three mutations that occur in >10% of breast carcinomas (those in P53, GATA3 and PIK3CA) and also few genes that are amplified in >10% of cases (including HER2, cyclinD1 and CMYC). There are hundreds of mutations that each occur at low frequency in breast cancer.

Gene expression profiling has led to the identification of subsets of breast cancer revealed by unsupervised classification termed basal type, ERBB2 like, luminal A, luminal B and normal epithelial like cancers; and supervised classification has revealed good- and poor prognosis subtypes. A growing number of prognostic tests based on gene expression profiling is used clinically. While identification of prognostic gene expression profiles has been successful, it has not been possible yet to identify robust clinically useful predictors of response to systemic treatment (chemotherapy, hormonal therapy, targeted therapy, checkpoint inhibitor immunotherapy).

Integration of histologic, genomic and gene expression data of breast carcinomas is leading to an increasingly refined classification that elucidates the initiation and progression of breast cancer at the molecular level; and the identification of novel prognostic and predictive markers that can guide treatment of individual patients.

Background reading:

M.J. van de Vijver, Molecular tests as prognostic factors in breast cancer, Virchows Arch. 2014 Mar;464(3):283-91
The molecular pathology of ovarian cancer

David Huntsman
UBC / BC Cancer Agency, Canada

Content not available at the time of printing.
The molecular pathology of endometrial cancer

Britta Weigelt
Memorial Sloan Kettering Cancer Center, USA

The uterine corpus represents the most common site for gynecologic malignancies in the western world. Endometrial cancer comprises a heterogeneous group of tumors with distinct risk factors, histopathologic features and clinical outcomes. Genomic studies are continuing to unveil the constellation of genetic alterations in uterine cancer, which have the potential to be used as molecular markers for classification, risk-stratification and therapy decision-making.

This presentation will focus on the molecular classification of endometrioid and serous endometrial carcinomas, including the different types of hypermutated cancers, and the advances in the genetic characterization of rare subtypes of the disease, including uterine clear cell carcinomas, adenosarcomas and carcinosarcomas. Furthermore, an update on the genetic underpinnings of endometrial stromal sarcomas will be presented. The limitations of the current classification systems and the challenges for the development of a taxonomy for endometrial cancer that accurately reflects its molecular characteristics and clinical behavior will be discussed.

Background reading:


The molecular pathology of melanoma

Daniel Peeper
NKI, Netherlands

For a long time, advanced-stage melanomas were refractory to the available therapeutic options, but recent developments have begun offering better perspectives for patients. The small molecule inhibitor vemurafenib, specifically targeting the mutant BRAFV600E kinase, was the first standard of personalized care for patients diagnosed with mutant BRAF metastatic melanoma. Although this compound initially reduces tumor burden dramatically, eventually most melanomas become resistant and progress on treatment. This occurs by the acquisition of additional mutations or other alterations, most of which reactivate the mitogen-activated protein kinase (MAPK) pathway. Although further suppression of BRAF-MAPK signaling by the inclusion of MEK inhibitor delays resistance, eventually most patients relapse.

The clinical outcome of late-stage melanoma patients has also greatly improved thanks to the recent availability of T cell checkpoint modulation, primarily by CTLA-4 and PD-1/PD-L1 blockade. But still, large patient groups fail to (durably) benefit from these treatments, underscoring the continuing need for developing novel therapeutic modalities.

Therefore, in spite of these new perspectives, there is a dire need to identify additional targets amenable to therapeutic intervention, possibly to be used in combination settings with tumor inhibitors alongside immune activators. We are studying (lack of) sensitivity to both tumor and immune cell treatment using patient biopsies, patient-derived xenografts (PDX) and low-passage cell lines. These systems are used for systematic function-based genetic screens to identify melanoma and immune cell factors representing pharmacologically tractable therapeutic targets. The results from these and related studies will be discussed.

Background reading:


The molecular pathology of colorectal cancer

Andreas Jung

LMU Munich, Germany

Content not available at the time of printing.
The molecular pathology of GISTS

Brian Rubin
Cleveland Clinic/Lerner Research Institute, USA

This lecture will focus on more recent developments related to gastrointestinal stromal tumor (GIST). GISTs were originally thought to harbor either KIT or platelet-derived growth factor receptor A (PDGFRA) mutations only, which are targeted by KIT and PDFRA inhibitors such as imatinib mesylate therapeutically. However, more recent discoveries have highlighted additional, less common oncogenic driver mutations including NF1, BRAF and succinate dehydrogenase (SDH) mutations, and gene fusions. Some of these newly discovered mutations are germline mutations which further complicate GIST patient management. Genotyping GISTs has become more important since not all genotypes respond equally to FDA-approved tyrosine kinase inhibitors. Because it is apparent that GIST is comprised of a family of related cancers driven by different oncogenic mechanisms, GIST has become a paradigm for personalized cancer therapy. Recent developments in GIST immunohistochemistry (IHC) demonstrate how IHC can be used to diagnose GIST and screen for specific GIST mutations. DOG1 is particularly useful in the diagnosis of KIT IHC negative GIST including those GISTs with PDGFRA mutations, which can also potentially be identified by PDGFRA immunohistochemistry. SDHB immunohistochemistry is useful in characterizing GISTs with SDHA-D mutations while SDHA immunohistochemistry is able to identify SDHA mutant GISTs. Finally, allele-specific BRAF V600E antibodies are useful in identifying BRAF V600E-mutated GIST.

Background reading:


The molecular pathology of bone tumours

Judith Bovee
Leiden University Medical Center, Netherlands

Bone tumours are considered difficult by most pathologists, as they are rare, have overlapping morphology, need radiological correlation, and the usefulness of immunohistochemistry is limited. Therefore, conventional morphology is still the cornerstone of the diagnosis. Over the past decade, more knowledge has become available on the molecular background of bone tumours. In sarcomas, we recognize three molecular classes of bone tumours. First, tumors with deregulated transcription, which is usually due to a translocation in which the fusion product acts as an aberrant transcription factor, include for instance Ewing sarcoma. Second, deregulated signalling can be caused by specific amplification (e.g. MDM2 in low grade osteosarcoma), specific gene mutation (e.g. GNAS mutation in fibrous dysplasia) or a translocation causing a promoter swap leading to upregulation of a specific gene (e.g. USP6 rearrangement in aneurysmal bone cyst or GRM1 rearrangement in chondromyxoid fibroma). Third, the largest subgroup includes sarcomas with genetic instability and complex karyotypes. These include osteosarcoma and high grade chondrosarcoma. Technical advancements including next generation sequencing have revealed many new genetic alterations in rare bone tumours over the past few years, which helps us to understand their histogenesis, may assist in the differential diagnosis and may provide targets for novel therapeutic strategies.

Background reading:


PA, Stratton MR, Campbell PJ, Flanagan AM., Distinct H3F3A and H3F3B
driver mutations define chondroblastoma and giant cell tumor of bone. Nat
Genet. 2013 Dec;45(12):1479-82. doi: 10.1038/ng.2814. Epub 2013 Oct

5. Mariño-Enríquez A, Bovée JV., Molecular Pathogenesis and Diagnostic,
2016 Sep;9(3):457-73.

6. van IJzendoorn DG, de Jong D, Romagosa C, Picci P, Benassi MS,
Gambarotti M, Daugaard S, van de Sande M, Szuhai K, Bovée JV., Fusion
events lead to truncation of FOS in epithelioid hemangioma of bone. Genes
Chromosomes Cancer. 2015 Sep;54(9):565-74
The molecular pathology of soft tissue sarcomas

Matt van der Rijn
Stanford School of Medicine, USA

In this talk I will present the use of a molecular approach to the diagnosis of soft tissue sarcoma. These are malignant tumors that originate from connective tissue cells such as muscle cells, fibroblasts and adipocytes. The disease is rare with approximately 11,000 new cases per year in the United States. In Europe, there is an annual incidence of 5.6 cases per 100,000 adults. Within this group of tumors there are over 50 distinct diagnostic entities. As a result most clinicians only rarely see cases from each subtype leading to unfamiliarity with treatment options but also with diagnostic classifications. Accurate diagnosis is of course a prerequisite for appropriate therapy and this is especially the case when one considers that novel targeted therapies are continuously being developed. Traditionally, classification of sarcomas has been based on the morphologic recognition these tumors and was supported by immunohistochemistry studies and a relatively limited set of molecular tests. In recent years the number of molecular changes that are characteristic for a particular tumor type has increased dramatically.

It has been recognized that on a molecular level, two broad categories of sarcoma can be identified. One group of sarcomas is characterized by highly complex genetic abnormalities in which to date no specific patterns can be recognized. Members of this category include leiomyosarcomas, undifferentiated pleomorphic sarcomas and malignant peripheral nerve sheath tumors. The second group of sarcomas has simple genetic changes that consist of chromosomal translocations, gene amplifications, and oncogenic mutations. Many of these simple genetic changes are relevant to the diagnosis of these tumor types as they occur specifically in only one tumor type. In addition they form the basis for much of the targeted therapeutic approaches that are in practice or are being developed.

Specific chromosomal translocations have been identified for more than 30 soft tissue sarcomas and this number can be expected to increase. It is not cost effective to maintain a set of individual diagnostic tests (either by RT-PCR or by FISH) for these rare disease in a CLIA-approved manner in diagnostic molecular laboratories. A number of NGS-based approaches have recently been developed that allow for the use of a single test to detect multiple translocations.

Background reading:

3. West et al. A landscape effect in tenosynovial giant-cell tumor from
activation of CSF1 expression by a translocation in a minority of tumor cells. PNAS, 2006, 103: 690-5


The molecular pathology of lung cancer

Marc Ladanyi
Memorial Sloan Kettering Cancer Center, USA

Content not available at the time of printing.

CTCs: a multi-use liquid biopsy

Caroline Dive
CRUK Manchester Institute, UK

Content not available at the time of printing.
The molecular pathology of the central nervous system tumours

Pieter Wesseling
VU University Medical Center, Netherlands
Princess Máxima Center for Pediatric Oncology, Utrecht, Netherlands
University Medical Center Utrecht, Utrecht, Netherlands

Changes in the WHO Classification of CNS tumors, i.e. the worldwide accepted standard for the diagnosis of these neoplasms, has important consequences as it serves as a guide for the design of studies monitoring response to therapy and clinical outcome. The revised 4th edition of this classification that was published in 2016 represents a paradigm shift as, for the first time, the definition of multiple CNS tumor entities is now partly based on particular genotypic characteristics. This is especially true for gliomas and embryonal CNS tumors. Additionally, compared to the original 4th edition (published in 2007), some splitting and lumping of entities occurred because of new insights in the genetic underpinnings of CNS tumors and/or based on more recently published clinico-pathologic studies.

A series of short reviews published in Neuropathol Appl Neurobiol in 2018 summarizes what has changed in the revised 4th edition of the WHO Classification of CNS tumors [2-4]. Additionally, these reviews provide:

- schematic representations of what the classification of gliomas (including mixed neuronal-glial neoplasms) and embryonal tumors looks like right now
- tables summarizing the most relevant molecular markers for the diagnosis of CNS tumors and the tools that can be used to assess these markers
- information on some diagnostic challenges and new perspectives.

As can be expected, after publication of the WHO 2016 Classification, further elucidation of the molecular underpinnings of CNS tumors continues at a rapid pace. A consortium with the acronym cIMPACT-NOW (‘consortium to Inform Molecular and Practical Approaches to CNS tumor Taxonomy – Not Officially WHO’) has been established in 2017 to determine how the most important, clinically relevant new information can be rapidly and practically incorporated into CNS tumor classification [5]. Indeed, this consortium has already published two reports in 2018 with guidelines on how to optimally use e.g. terms and (novel) molecular markers in clinical practice (check PubMed for ‘cIMPACT-NOW’).

In line with the contents of the reviews mentioned above [2-4], the lecture on the molecular pathology of CNS tumors in the 8th EACR-OECI course will highlight

- CNS tumors where molecular diagnostics plays an important role;
- the clinically most relevant molecular markers;
- tools that may be used to assess the status of these markers in CNS tumors;
- some pitfalls in molecular diagnostics of CNS tumors;
- some promising new developments.
Background reading:


The evolution of pathology

Giorgio Stanta
University of Trieste, Italy

Pathology is rapidly evolving and today clinical research (research based on patients’ biological material) is going to be an integral part of the clinical activity. Only clinical material can help us define the very wide range of clinical heterogeneity. This is also connected with new types of clinical studies such as N-1 trials and basket, umbrella and platform studies. This is because individual tumours considerably vary and exceed the variation between distinct cancer types. The two main goals of diagnostics and clinical research in oncology are to avoid recurrences by predicting a correct adjuvant therapy after surgery and, in this case, tumour tissues are used with extractive and in-situ methods. The second goal is to control recurrences by target therapy or immunotherapies. In this case, liquid biopsies are going to be the major clinical references.

The major problem in this kind of research is reproducibility, which is related to many distinct technical reasons that are actively studied in many European organizations and projects. Intratumour heterogeneity is one of the main problems not only in research but also for clinical application. A new type of approaches should be studied and standardized.

Background reading:

5. Stanta G. Tissue Heterogeneity as a Pre-analytical Source of Variability. Recent Results Cancer Res. 2015;199:35-43.
Conflict of Interest Disclosure Form

(to be completed by scientific programme committee members)

NAME: Leonor David

AFFILIATION: IPATIMUP and Medical Faculty of the University of Porto

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Stock shareholder:

Spouse/partner:

Other support (please specify):

Signature: 

Date: 27/10/2017

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NAME: Richard Morris

AFFILIATION: Cancer Research UK Manchester Institute

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Spouse/partner:

Other support [please specify]:

Institute of Cancer Research Rewards to Inventors Scheme

Signature: Richard Morris

Date: 03/11/2017
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NAME: Jorge Sergio Reis-Filho

AFFILIATION: Memorial Sloan Kettering Cancer Center

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Signature: [Signature]
Date: 11/24/2017
Conflict of Interest Disclosure Form
(to be completed by scientific programme committee members)

NAME: Giorgio Stanta

AFFILIATION: University of Trieste

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Stock shareholder:

Spouse/partner:

Other support (please specify):

Signature: ____________________________

Date: 8th February 2018
Conflict of Interest Disclosure Form

(to be completed by scientific programme committee members)

NAME: Marc J. van de Vijver

AFFILIATION: Academic Medical Center, Department of Pathology, Amsterdam

In accordance with criteria 14 of document EUMS 2016/20 "EACCME® criteria for the Accreditation of Live Educational Events (LEE)", all declarations of potential or actual conflicts of interest, whether due to a financial or other relationship, must be provided to the EACCME® upon submission of the application. Declarations also must be made readily available, either in printed form, with the programme of the LEE, or on the website of the organiser of the LEE. Declarations must include whether any fee, honorarium or arrangement for reimbursement of expenses in relation to the LEE has been provided.

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Name of commercial company

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Hoffmann La Roche Ltd

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Member Pathology Advisory Board Hoffmann La Roche Ltd

Member Pathology Advisory Board Merck, Sharp and Dohme

Member Advisory Board Genomic Health

Co-inventor 70-gene prognosis profile in breast cancer

Signature: [Signature]

Dates: 23 November 2017
The organisers wish to express their appreciation for the support provided by sponsors at the 8th EACR-OECI Joint Course: Molecular Pathology Approach to Cancer. Their interest and enthusiasm for the course has enabled the organisers to provide an impressive scientific programme.

### Exhibitors

![Exhibitor Logos](image)

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8th EACR-OECI Joint Course: Molecular Pathology Approach to Cancer
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Notes
Notes
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The OECI is a non-governmental, non-profit Organisation founded in Vienna in 1979 and remodelled in 2005 into OECI-EEIG, a European Economic Interest Grouping, headquartered in Brussels.

Today, the OECI counts 88 Member Institutes, which include some of the most prominent European Comprehensive Cancer Centres.

The OECI aims to create a critical mass of expertise and competences, contributing to the production and dissemination of knowledge, so as to reduce fragmentation and increase competitiveness. These goals are being achieved by promoting and strengthening the concept of “comprehensiveness”, supporting quality in cancer care also through a well-structured internal organisation.

OECI aims to accelerate the production and application of personalised care approaches, and to ensure equal rights to all cancer patients, with the ultimate goal of finding new and better treatments, providing more comprehensive care and improving patient quality of life, through evidence-based medicine.

The increasing interest from international organisations, stakeholders & cancer community in the OECI points to the growing importance of a comprehensive cancer network of institutions, where the entire chain of cancer care provision is present.

The strong alliance with the European Cancer Patient Coalition – ECPC, provides a coherent picture of today’s cancer patients’ expectations, ranging from: quality of care to information on survivorship and medical nutrition, improvement in the quality of interdisciplinary patient treatment, harmonisation in oncology healthcare practices and patient advocacy capacity to effectively tackle cancer care inequalities in Europe.

Giving the crucial role to pathology departments in oncology and in lieu of the expected “influx” of new markers and diagnostics, the OECI acts in close cooperation with the European Association for Cancer Research and the European Society of Pathology, in order to better disseminate the innovation process amongst its members and abroad. The EACR-OECI training course series on “Precision Medicine” and “Molecular Pathology Approach to Cancer” in Amsterdam, are just two examples of the OECI efforts to promote the innovation in cancer diagnosis and care.
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