SUMMER SCHOOL IN TRANSLATIONAL CANCER RESEARCH 2021

13 - 14 October

Virtual Event

Cancer Core Europe

Register online (free of charge): https://indico.dkfz.de/e/summerschool21

Wednesday, 13th

Chair	Prof. Stefan Fröhling (National Center for Tumor Diseases (NCT) Heidelberg, Germany)
01:00 p.m.	**Welcome and Introduction** **Prof. Emile Voest** (Chairman of Board of Directors CCE; The Netherlands Cancer Institute (NCI), Amsterdam) **Prof. Stefan Fröhling** (National Center for Tumor Diseases (NCT) Heidelberg, Germany)
01:10 p.m.	**Cancer Prevention: Implementation and how to measure success** **Dr. Joachim Schüz** (Cancer Prevention Europe; International Agency for Research on Cancer, France)
01:55 p.m.	**Immunotherapy / Microbiome: Microbiota as a new path to treat breast cancer** **Dr. Elda Tagliabue** (Instituto Nazionale dei Tumori (INT) Milano, Italy)
02:35 p.m.	**Short Break & Chat**
Chair	Dr. Vasilios Zachariadis (Karolinska Institutet, Stockholm, Sweden)
02:45 p.m.	**Flash Talk: Mechanisms of hyperprogressive disease in lung cancer patients and tumor models** **Dr. Roberto Ferrara** (Istituto Nazionale dei Tumori, Milano, Italy)
02:55 p.m.	**Flash Talk: Somatic versus germline genetics** **Dr. Emma Tham** (Karolinska Institutet, Stockholm, Sweden)
03:05 p.m.	**Flash Talk (title tbd)** **Dr. Bruno Köhler** (Heidelberg University Hospital, NCT Heidelberg, Germany)
03:20 p.m.	**Interactive Format**
Chair	Prof. Svetlana Bajalica Lagercrantz (Karolinska Institutet, Stockholm, Sweden)
04:15 p.m.	**The EMBO Keynote Lecture:** **Hallmarks of Cancer - 21st Anniversary / Beyond Hallmarks of Cancer** **Prof. Douglas Hanahan** (École Polytechnique Fédérale de Lausanne (EPFL), Switzerland)
05:00 p.m.	**Genomics research meets patients** **Dr. Corrie Painter** (Broad Institute of Massachusetts Institute of Technology (MIT) and Harvard, USA)
05:40 p.m.	**Closing Remarks Day 1**
06:00 p.m.	**Optional Workshop (Scientific Writing, Grant Writing)** **Dr. Angelika Hofmann** (SciWri Services and Dartmouth College, USA)

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Cancer Core Europe

dkfz. German Cancer Research Center in the Helmholtz Association

Research for a Life without Cancer

ORGANIZED BY

NCT National Center for Tumor Diseases Heidelberg

supported by German Cancer Research Center (DKFZ) Heidelberg University Medical Center Hospital for Thoracic Diseases German Cancer Aid

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EMBO excellence in life sciences

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Cancer Prevention Europe

SUMMER SCHOOL IN TRANSLATIONAL CANCER RESEARCH 2021

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Thursday, 14th

Chair	Dr. María Abad (Vall d'Hebron Institute of Oncology (VHIO), Barcelona, Spain)
01:00 p.m.	**Welcome and Introduction** **Dr. María Abad** (Vall d'Hebron Institute of Oncology (VHIO) Barcelona, Spain)
01:10 p.m.	**The Mutational Foot Prints of Cancer Therapies** **Prof. Núria López-Bigas** (Institute for Research in Biomedicine (IRB) Barcelona, Spain)
01:55 p.m.	**The Next Generation of Precision Medicine Trials** **Dr. Richard Baird** (Cancer Research UK (CRUK) Cambridge Centre, UK)
02:35 p.m.	**Short Break & Chat**
Chair	Dr. Dr. Andreas Mock (National Center for Tumor Diseases (NCT) Heidelberg, Germany)
02:45 p.m.	**Flash Talk: Cancer immunotherapy – Exploiting innate memory in solid tumors** **Dr. Dhifaf Sarhan** (Karolinska Institutet, Stockholm, Sweden)
02:55 p.m.	**Flash Talk (title tbd)** **Dr. Irene Braña** (Vall Hebron Institute of Oncology (VHIO), Barcelona, Spain)
03:05 p.m.	**Flash Talk: New Module of CCE Basket of Baskets (BoB) trial** **Speaker tbd: winner of the call for BoB module Ideas 2021**
Chair	Prof. Ingemar Ernberg (Karolinska Institutet, Stockholm, Sweden)
03:20 p.m.	**Aging and Metabolism, Cellular Plasticity and Disease** **Prof. Manuel Serrano** (Institute for Research in Biomedicine (IRB) Barcelona, Spain)
04:05 p.m.	**A Personal History about Hereditary Diffuse Gastric Cancer CDH1** **Dr. Tanya Bisseling** (Radboud University Medical Center, The Netherlands)
04:45 p.m.	**Closing Remarks**
05:00 p.m.	**Case Study Discussion / Molecular Tumor Board Simulation** **PD Dr. Dr. Peter Horak, Dr. Dr. Andreas Mock, Dr. Marcus Renner, Dr. Maria-Veronica Teleanu** (National Center for Tumor Diseases (NCT) Heidelberg and Heidelberg University Hospital, Germany)

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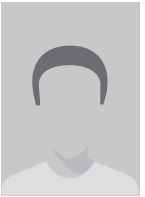


Dr. Richard Baird

Cancer Research UK (CRUK) Cambridge Centre, UK

The Next Generation of Precision Medicine Trials

Dr. Tanya Bisseling

Radboud University Medical Center Nijmegen,
the Netherlands

A personal history about hereditary diffuse gastric cancer CDH1

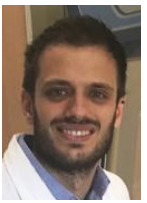
A CDH1 pathogenic germline variant is very rare, estimated 0,2-0,6/100.000. As a result, this variant is easy to overlook. Unfortunately, an individual with this mutation has about 70% life time risk to develop diffuse type stomach cancer and a woman had a 40-60% risk to develop lobular breast cancer. As such, for an individual the consequences of a carriership are huge. Therefore, recognition of families with carriership of hereditary diffuse gastric cancer is important.

This presentation gives insight into the basic hereditary information, clinical aspects an personal patient experiences. Goal is to make sure you'll never forget CDH1!

Dr. Irene Brana

Vall d'hebron Institute of Oncology (VHIO), Barcelona, Spain

Flash Talk



Dr. Roberto Ferrara

Medical Oncology Unit
Molecular Immunology Unit
Fondazione IRCCS Istituto Nazionale dei Tumori di Milano

Mechanisms of hyperprogressive disease in lung cancer patients and tumor models

Circulating immune determinants of hyperprogressive disease (HPD) in non-small cell lung cancer (NSCLC) patients treated with PD-1/PD-L1 inhibitors (ICI) alone or in combination with platinum-based chemotherapy (PCT)

Background. HPD has been described in ≃14-26% of NSCLC patients upon single-agent ICI and has not been reported upon ICI and PCT combinations. Both high circulating neutrophils and senescent T-cells correlated with HPD, however the neutrophils-T-cells interplay and the role of specific neutrophils subsets in driving HPD is unknown.

Methods. NSCLC patients treated with 1st line ICI as single-agent or in combination with PCT were assessed for HPD and circulating neutrophils' phenotype. HPD required 3 assessment (2 before ICI, 1 upon ICI) and was defined as delta tumor growth rate (TGR) (TGR upon ICI – TGR before ICI) >50% and/or TGR ratio (TGR upon ICI/ TGR before ICI) ≥2. Circulating low density neutrophils (LDNs) subtypes were assessed by flow cytometry on peripheral blood mononuclear cells (PMBCs) and defined as CD66b+CD15+ cells among CD11b+ PBMCs. Immature subtypes were defined as CD10- LDNs. T-cells were isolated from healthy donors and cocultured with patients' LDNs to characterize the neutrophils-T-cells interplay. LDNs subtypes were isolated from patients and treated in-vitro with cisplatin to assess cell death.

Results. 46 NSCLC patients were treated with single-agent ICI and 17 with PCT+ICI. In the ICI single-agent cohort, PD and HPD occurred in 21 (41%) and 4 (9%) patients. Before ICI start, HPD patients had significantly higher median % of circulating immature CD10- LDNs neutrophils [43.5 (min 29.5; max 82.6) vs 10.3 (min 0.1; max 79.4), p=0.01] compared to PD patients.

In the ICI-PCT cohort no HPD was reported. 5 patients had baseline CD10- LDNs ≥ 43.5% (median % of CD10- LDNs in HPD patients upon single-agent ICI). In these 5 patients, CD10- LDNs significantly decreased during ICI-PCT compared to what observed in HPD patients upon single-agent ICI [median variation -43.4 (min -67.6, max -31.6) vs +6.9 (min -33, max +44), p= 0.03]. After 7 days of coculture, immature CD10- LDNs significantly reduced T-cells survival and promoted a T-cell senescent phenotype (CD28 loss, CD57 gain) impairing T-cells proliferation and increasing IFN-gamma production. Cisplatin treatment significantly increased cell death among CD10- LDNs compared to CD10+ LDNs.

Conclusion. Higher baseline immature CD10- LDNs impair T-cell survival and promote T-cell senescence being a circulating biomarker of HPD upon single-agent ICI. The addition of PCT prevents HPD by inducing immature neutrophils cell death.

Prof. Douglas Hanahan

Ludwig Institute for Cancer Research, Lausanne Branch
Swiss Federal Institute of Technology Lausanne (EPFL), Switzerland
Swiss Cancer Center Leman

The EMBO Keynote Lecture: Hallmarks of Cancer - 21st Anniversary / Beyond Hallmarks of Cancer

The hallmarks of cancer constitute an organizing concept that may provide a rational basis for distilling the diversity and complexity of human cancers so as to better understand mechanisms of the disease and its manifestations (Hanahan and Weinberg, 2011). The conceptualization involves eight acquired capabilities – the hallmarks of cancer - and two generic characteristics of neoplastic disease that facilitate their acquisition during the multistage process of neoplastic development and malignant progression. These capabilities consist of sustaining proliferative signaling, evading growth suppressors, resisting cell death, enabling replicative immortality, inducing/accessing vasculature, activating invasion and metastasis, deregulating cellular energetics and metabolism, and avoiding immune destruction. The principal facilitators of their acquisition are genome instability with consequent gene mutation, and tumor-promoting immune inflammation. The integration of these hallmark capabilities in symptomatic disease involves multiple cell types populating the tumor microenvironment, including heterogeneous populations of cancer cells, in particular cancer stem cells, and three prominent classes of stromal support cells – angiogenic vascular cells, cancer associated fibroblasts, and infiltrating immune cells. Notably, these stromal cells have the demonstrated capacity to contribute to seven of the eight hallmark capabilities (Hanahan and Coussens, 2012). Thus, while the functional contributions of stromal cells and their pathologic importance will likely vary between different cancers and indeed at different stages of tumorigenesis and tumor progression, the evidence is clear that a sole focus on the transformed cancer cell (and its genome) cannot fully inform us about mechanisms of the disease. One premise to be discussed is that the hallmarks of cancer may prove to be a useful heuristic tool for designing innovative new mechanism-guided (hallmark-targeting) therapeutic approaches for cancer treatment. A second is the possibility that there are additional emerging parameters of the conceptualization that merit discussion, debate, and experimental elaboration.

Dr. Bruno Köhler

Heidelberg University Hospital, NCT Heidelberg,
Germany

Bcl-xL is an oncogenic driver in gastrointestinal tumors

Introduction. The evasion of cell death is a classical hallmark of cancer cells. Classical programmed cell death, termed apoptosis, is a tightly regulated program of cellular suicide. The balance between antiapoptotic (Mcl-1, Bcl-2 and Bcl-xL) and proapoptotic Bcl-2 proteins is crucial for survival. Malignant transformation is often concomitant with a deregulated cell death machinery, promoting tumor growth. In the therapeutic setting, an increased antiapoptotic signaling has been shown to mediate primary and acquired therapy resistance. Based on these broad implications for tumor biology, targeting antiapoptotic proteins harbor great potential. Equipped with highly specific Bcl-2 protein inhibitors, we embark on experiments to explore their translational value in gastrointestinal tumors. **Methods & Results.** Our group harnesses cutting-edge in vitro, in vivo and ex vivo methodology to pave the way for a future clinical trial application of specific Bcl-2 protein inhibition. We use 3D cell culture, organoids, human tissue cultures, and genetic engineered mouse models to study expression patterns and vulnerabilities in the cell death signaling networks. International collaborations enable the investigation of large cohorts with comprehensive clinical annotation on the DNA, RNA and protein level to understand a possible prognostic and predictive significance of antiapoptotic proteins. Our data identified Bcl-xL as being of dominant importance, compared to Bcl-2 and Mcl-1, in a variety of solid tumors including colorectal cancer and cholangiocarcinoma. Compared to other antiapoptotic proteins, such as Bcl-2 and Mcl-1, Bcl-xL showed higher expression as well as increased protein activity. In vitro and in vivo targeting of Bcl-xL induced cell death and sensitized resistant tumor cells towards standard therapy (e. g. chemotherapy and irradiation). **Conclusion & Perspective.** In contrast to hematological malignancies, Bcl-xL is the most relevant Bcl-2 protein in numerous solid tumors. Thereby, Bcl-xL might be both – driving force and Achilles´ heel. Moving towards clinical translational, stratification based on expression patterns might identify patients who benefit most from a Bcl-xL inhibition in combination with standard of care.

Prof. Nuria Lopez-Bigas

Institute for Research in Biomedicine (IRB) Barcelona,
Spain

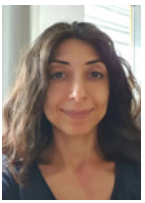
Computational analysis of cancer genomes

Somatic mutations are the driving force of cancer genome evolution. Given the evolutionary principles of cancer, one effective way to identify genes involved in cancer is by tracing the signals left by the positive selection of driver mutations across tumours. We analyze thousands of tumor genomes to generate a compendium of cancer genes across tumor types (http://www.intogen.org). Most mutations identified in tumors in cancer genes are mutations of unknown significance. The mutations observed in thousands of tumors, --natural experiments testing their oncogenic potential replicated across individuals and tissues-- may be exploited to identify driver mutations in cancer genes. From these mutations, we extract features that describe the mechanism of tumorigenesis of each cancer gene and tissue and use those to build machine learning models that effectively identify driver mutations. With those models we perform in silico saturation mutagenesis to outline blueprints of potential driver mutations in cancer genes. These blueprints support the interpretation of newly sequenced patients' tumors and the study of the mechanisms of tumorigenesis of cancer genes across tissues. The analysis of tumor cohorts provides valuable information to improve the interpretation of individual variants detected in newly sequenced tumors in clinical or research settings. We have developed CancerGenomeInterpreter.org, a tool designed to identify driver mutations and biomarkers of drug response in individual tumors.

Dr. Corrie Painter

Broad Institute of Massachusetts Institute of Technology (MIT) and Harvard,
USA

Genomics research meets patients



Dr. Dhifaf Sarhan

Karolinska Institutet, Department of Laboratory Medicine,
Division of Pathology, Stockholm,
Sweden

Cancer immunotherapy- Exploiting innate memory in solid tumors

Natural Killer (NK) cells are innate immune cells able to reject hematological malignancies and correlate with better prognosis in solid tumors. However, conventional NK (cNK) cell anti-tumor activity is limited by the immune suppressive tumor microenvironment (TME). We have recently discovered that an adaptive NK (aNK) cell subpopulation with immunological memory is able to resist immune suppression. Therefore, given their capacity to resist TME suppression and kill tumors, aNK cells provide an excellent new approach for cancer immunotherapy. Here, we found that aNK cells become tumor reactive to melanoma primary tumor cell lines following 10 days of culture with dendritic cells (DC) loaded with melanoma tumor lysate but do not react to irrelevant tumor cells. In melanoma and serous ovarian cancer, we found that tumor infiltrating aNK cells were able to recognize autologous tumors and elicit recall/memory responses in form of degranulation (CD107a) and cytokine production compared with their counterpart cNK cells both after short-term (10 days) and long-term (4 weeks) restimulation compared to allogeneic tumors, supporting our hypothesis that aNK cells possess specific recognition of tumor antigens. We also found that such tumor reactivity was correlating with clinical responses to anti-PD1 immune checkpoint therapy in melanoma patients. Notably, aNK cells were located in the center of pancreatic tumors compared to cNK cells located in the stroma. We next investigated aNK cell prognostic value in different solid tumors. Data from public databases were utilized to overlay our used aNK cell phenotype signature with the clinical responses. We found that high aNK cell gene signature was correlating with improved survival in skin cancer, ovarian, pancreatic, and breast invasive carcinoma patients. Altogether, these observations suggest a predictive value of aNK cells patients with solid tumors and highlight their therapeutic potential to serve as immunotherapy.

Dr. Joachim Schüz

International Agency for Research on Cancer,
World Health Organisation (IARC/WHO),
Environment and Lifestyle Epidemiology Branch, Lyon, France

Cancer Prevention: Implementation and how to measure success

The burden of cancer is increasing worldwide. While the estimated total number of new cancer cases (excluding non-melanoma skin cancers) was 14.1 million in 2012, it has been estimated to be 18.1 million for 2020 (an increase of about 28%) and projected to further increase to 28.0 million in 2040 should this trend not be stopped or reversed. Time trends of cancer occurrence are monitored and reported by the Global Cancer Observatory of the IARC/WHO, by cancer site and region, showing that the strongest increases between 2020 and 2040 are predicted for Africa (89%), Latin America and Caribbean (64%), Asia (59%) but still substantial for Oceania (49%), North America (31%) and Europe (19%). Even the wealthiest of countries do not have the capacities to treat their way out of such an increasing cancer burden, strengthening the need for more rigorous primary and secondary cancer prevention. For Europe, it has been suggested that one third to half of all cancer cases are preventable, as most of the established causes are exposures (including chemical, physical or biological agents) or unhealthy behaviours that are modifiable. Scientific evidence has been translated into a set of public health recommendations targeted to the individual summarising of what they can do themselves to reduce their risk of cancer, called the 'European Code against Cancer', which remains a key cancer prevention tool in the recently launched Europe's Beating Cancer Plan. Adaptions are under development for other regions of the world, notably for Latin America and the Caribbean. This presentation provides an overview of the present cancer burden, cancer prevention recommendations and their implementation, and indicators used to monitor time trends and successes of prevention strategies, and how they are calculated.

Dr. Manuel Serrano

Group Leader
Institute for Research in Biomedicine (IRB) Barcelona, Spain
ICREA Professor

Cellular senescence as a novel target for cancer therapy

Cellular senescence is a response to damage characterized by a stable cell cycle arrest and a potent secretion of proliferative, inflammatory and matrix remodelling factors. While the activation of senescence in tumor cells disables their proliferative capacity, the local microenvironment that they generate favours the regrowth of residual therapy-surviving cancer cells. The therapeutic potential of senescence-targeting strategies has been established in proof-of-concept studies, but clinically-relevant evidences are still lacking. A particularly exciting approach combines standard-of-care cancer therapeutics, that often trigger cellular senescence, with novel agents known as "senolytics" that kill these senescent cells. Currently, only a handful of senolytic targets are known and there are few senolytic experimental compounds. I will present our recent work on targeting senescence to improve standard cancer therapy.

Dr. Elda Tagliabue

Molecular Targeting Unit, Dept of Research,
Fondazione IRCCS Istituto Nazionale dei Tumori of Milan,
Italy

Microbiota as a new path to treat breast cancer

It is becoming increasingly clear that the development, growth, and health of macroorganisms are influenced by the microbial communities they host. These communities including commensal, symbiotic, and pathogenic microorganisms (bacteria, viruses and fungi) that share our body space make up the microbiota. Recently, next-generation nucleic acid sequencing led us to obtain an accurate fingerprint of the microbiota present in different body sites. The largest collection of these microorganisms is found in the gastrointestinal tract and is fundamental for several functions including food degradation, pathogen protection, vitamin production, immune system maturation and behavior. Recent studies have shown that gut microbiota composition can predict the risk of developing diseases and identification of microbiota signatures have started to be unraveled for early diagnosis, prognosis and response to therapy of cancers. In this context, our recent results revealed the impact of the gut microbiota on immune-mediated trastuzumab antitumor efficacy. In mice, the antitumor activity of trastuzumab was impaired by antibiotic administration or fecal microbiota transplantation from antibiotic-treated donors by impairing the recruitment of CD4+ T cells and GZMB+ cells as well as by reducing dendritic cell (DC) activation and the release of IL12p70 after trastuzumab treatment. Fecal microbiota β-diversity segregated patients according to response to neoadjuvant treatment containing trastuzumab and, similar to antibiotic-treated mice, positively correlated with immune signature related to interferon, IL12-NO, activated CD4+ T cells and activated DC in tumors, suggesting that manipulation of the gut microbiota is an optimal future strategy to achieve a therapeutic effect or to exploit its potential as a biomarker for treatment response.

In the last few years, there is emerging evidence that bacteria are present in human tumors and associate with different cancer hallmarks. Thus, better understanding of gut and tumor commensals and their relationship may pave the way for novel treatment options for cancer patients.

Dr. Emma Tham

Karolinska Institutet, Stockholm, Sweden

Somatic versus germline genetics

What is the difference between somatic variants, somatic mosaicism and germline variants?

Somatic variants arise in an adult cell, somatic mosaicism describes variants that arise in the post-zygotic embryo and germline variants are inherited from a parent or arose in the fertilised egg. Most of the latter are benign variants, but in rare cases may cause hereditary disease. **Which kinds of variants can be somatic?** All kinds of genetic variants can be somatic, i.e. single nucleotide variants, copy number variant, structural variants, epigenetic variants. **How do you know if a somatic variant is a true cancer driver?** It is important to consider the material analysed, the tumour cell purity, the method used, risk of artefacts (FFPE), which variants are normal variants and which are cancer variants (using databases of individuals without cancer; databases of cancer variants, databases with annotated variants, understanding the function of the gene and if a loss-of-function or gain-of-function variant is expected to perturb its function; the expected allele frequency in the cancer). **Why is it important to identify hereditary cancer syndromes?** The index patient and their relatives can be offered predictive genetic testing and carriers can be offered surveillance to reduce their risk of advanced cancer, leading to increased survival and decreased morbidity. In some cases prenatal testing such as pre-implantation genetic diagnosis may be possible. **How do you know if a germline variant causes a hereditary cancer syndrome?** Stringent criteria are used to identify true pathogenic variants and only genes and variants known to increase the risk of cancer are clinically actionable. The Department of Clinical Genetics are experts and can be consulted.

References: Standards and Guidelines for the Interpretation and Reporting of Sequence Variants in Cancer: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5707196/. Standards and Guidelines for the Interpretation of [Germline] Sequence Variants: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4544753/

Speaker tbd: winner of the call for BoB module Ideas 2021

Cancer Core Europe (CCE)

Flash Talk: New Module of CCE Basket of Baskets (BoB) trial

Basket of Baskets (BoB) is a large modular, open-label, phase II, multicentre study to evaluate targeted agents in molecularly selected populations with advanced solid tumours. The BoB trial contains several modules and is coordinated by Vall d'Hebron Institute of Oncology (VHIO) on behalf of Cancer Core Europe (CCE). The BoB team had recently launched a call for new BoB modules, the results of which will be presented here. For further information on BoB, please see https://bobtrial.cancercoreeurope.eu/

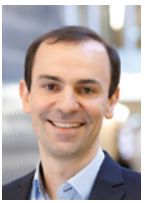
Dr. Angelika Hofmann

SciWri Services and Dartmouth College,
USA

Workshop Fundamentals of Scientific Writing: Publications and Proposals

Wednesday, October 13th, 2021, 6:00 - 7:20 p.m. CEST

This workshop aims to aid scientists in improving their science writing skills. The workshop will provide critical insights and practical advice on writing for scientific publications and grant proposals. It will cover select basic principles of scientific style and composition with the overall goal to achieve maximum impact with the reader and reviewer in mind.




PD Dr. Dr. Peter Horak, Dr. Dr. Andreas Mock, Dr. Marcus Renner, Dr. Maria-Veronica Teleanu

National Center for Tumor Diseases (NCT) Heidelberg, Heidelberg University Hospital (UKHD), German Cancer Research Center (DKFZ), Heidelberg, Germany

Workshop Case Study Discussion / Molecular Tumor Board Simulation

Thursday, October 14th, 2021, 5:00 - 6:30 p.m. CEST

This workshop aims to give registered participants an introduction on how clinical cases are discussed in a molecular tumor board. Participants can preview specific cases, check out necessary tools, and get a clearer picture of the process of considering and understanding molecular patient data. There will be a live demonstration as well as time for an interactive discussion. The Molecular Tumor Board is a well established structure at the NCT Heidelberg and experienced speakers will be discussing the cases and procedures with you.

(Due to limited capacities, this workshop is limited to participants who received a confirmation.)