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# 13<sup>th</sup> European Pathology Congress

August 02-03, 2017 Milan, Italy

# Keynote Forum DAY 1

# Euro Pathology 2017



# E Blair Holladay

American Society for Clinical Pathology, USA

### Cancer diagnostics in Africa: Leapfrog technologies to transform care

Nancer is a bigger problem in developing countries than human immunodeficiency virus (HIV)/acquired immunodeficiency /syndrome (AIDS). It is emerging as a leading cause of death in Sub Saharan Africa with a population close to one billion people. Moving cancer treatment to global health settings has been seen as costly, challenging or close to impossible. However, early detection may lead to more affordable and effective cancer treatment so new diagnostics technologies have the potential to help overcome global healthcare disparities for cancer. The solution lies in local physicians having access to a system that ensures rapid, accurate, and reliable pathology for primary diagnosis of cancer. Sub Saharan Africa faces immense challenges in providing adequate coverage and each region, country, and district with unique obstacles to overcome when meeting the health needs of the population. The American Society of Clinical Pathology (ASCP), in partnership with the Obama White House Office of Science Technology Policy and the Clinton Global Initiative, recently launched a 150 million dollar multi-year initiative which begins with assessment of potential countries with the greatest need--including collaboration and capacity program building with local officials and staff-to deploy full service pathology infrastructure for eligible countries to strategically meet their population needs. Working in parallel and together, steering committees for diagnostics and technology, care and treatment, in-country medical education, bioethics, and monitoring & evaluation have focused on each potential country to optimize success. The optimal solution includes the deployment of automated histopathology systems and integrated whole slide imaging systems linked through a customized laboratory information system to a dedicated team of pathologists from the United States. This long-term project will roll out to 20 or more countries in Africa as well as Haiti.

### **Biography**

E Blair Holladay serves as Chief Executive Officer of the American Society for Clinical Pathology (ASCP). He has focused on "Globalization initiatives for the medical laboratory community that include significant contributions to the President's Emergency Plan for AIDS Relief (PEPFAR) funded through the Centers for Disease Control and Prevention; strategic partnerships in laboratory medicine; corporate reorganization/management activities; mergers and acquisitions; international outreach; external partnerships; and health services research and delivery". ASCP has been funded for over \$40 million to support patient-centered care. His scientific research work focuses in the areas of Cytopathology and Molecular Research. He is a Principal Investigator for 50 scientific research grants and 100 scientific corporate contracts. He has published a number of significant research articles within the profession and is the primary author of several textbooks that are international best sellers. He is an active scientific Lecturer and has given over 170 national and international speeches.

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# Giuseppe Scalabrino

University of Milan, Italy

### Involvement of normal prions in some human myelin diseases

We have experimentally demonstrated that cobalamin (Cbl) deficiency increases normal cellular prion (PrPC) levels in rat spinal cord (SC) and cerebrospinal fluid (CSF), and decreases PrPC-mRNA levels in rat SC. Repeated intracerebroventricular administrations of anti-octapeptide repeat-PrPC-region antibodies to Cbl-deficient (Cbl-D) rats prevent SC myelin lesions, and the administrations of PrPCs to otherwise normal rats cause SC white matter lesions similar to those induced by Cbl deficiency. Cbl positively regulates SC PrPC synthesis in rat by stimulating the local synthesis of epidermal growth factor (EGF), which also induces the local synthesis of PrPC-mRNAs, and down-regulating the local synthesis of tumor necrosis factor (TNF)- $\alpha$ , thus preventing local PrPC overproduction. We have clinically demonstrated that PrPC levels are increased in the CSF of patients with sub-acute combined degeneration (SCD), unchanged in the CSF of patients with Alzheimer's disease and amyotrophic lateral sclerosis, and decreased in the CSF and SC of patients with multiple sclerosis (MS), regardless of its clinical course. We conclude that SCD (human and experimental) is a neurological disease due to excess PrPC without conformational change and aggregation, that the increase in PrPC levels in SCD and Cbl-D polyneuropathy and their decrease in MS CNS make them antipodean myelin diseases in terms of quantitative PrPC abnormalities, and that these abnormalities are related to myelin damage in the former, and impede myelin repair in the latter.

### **Biography**

Giuseppe Scalabrino Born in Milan, on July 4, 1944. He Studied in Institute of General Pathology, University of Milan from 1965 to 1968 and at 1968 he became M.D., magna cum laude, discussing an experimental thesis on the radiosensitizing properties of aliphatic aldehydes.

He worked in several positions as faculty of Institute of General Pathology at University of Milan from 1969 to present. He was the Associate Professor of General Pathology at University of Milan from 1971 to 1985. He was honored as Assistant to the Chairman of General Pathology, Faculty of Medicine and Surgery at University of Milan from 1973 to 1985. He has more than 100 Publications in high impact journals.

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# Maria Teresa Mascellino

Sapienza University of Rome, Italy

### Helicobacter pylori infection: Pathogenesis and therapeutic strategies

**Introduction & Aim:** *Helicobacter pylori* (Hp) are gram-negative mobile bacilli, difficult to be cultured, and able to cause different diseases. In fact, Hp is involved in chronic active gastritis, peptic ulcer disease, gastric carcinoma and mucosa-associated lymphoid tissue lymphoma (MALT) other than in endothelial dysfunction leading to vascular diseases. Hp is increasingly difficult to treat. The treatment regimens are declining in efficacy and the therapy of this infection is bedevilled by drug-resistant strains. Aim of our research is to study a population of 50 pangastritis already undergone multiple therapies and to evaluate the eradication rates.

**Methods:** All patients were positive to UBT (Urease Breath Test) then surely infected by Hp. Three biopsies were taken for each patient and submitted to rapid urease test, culture and antibiotics susceptibility by E-test.

**Results:** Out of 50 patients, culture and susceptibility testing were obtained in 31 patients (62%) whereas in 19 (38%) no *H. pylori* growth was detected. The first group was treated following the scheme shown in the image whereas the second one was empirically treated with antibiotics never taken before or with rescue therapies. The eradication rates were 52% and 63%, respectively.

**Conclusions:** No significant difference has been seen between the two groups. Probably the higher eradication rate in patients empirically treated, where no microorganisms have been isolated, can be due to the presence of bacteria not able to grow in culture then in a less virulent or dormant phase or in a very low number to be detected. Anyway, in our study the eradication rates of these pangastritis patients undergone multiple treatments are very low. The Toronto Consensus Group (2016) has proposed new treatment strategies recommending to prolong the cure from 10 to 14 days, to use bismuth quadruple therapy containing metronidazole and tetracycline as well as various rescue therapies.

#### **Biography**

Maria Teresa Mascellino has completed her MD in Rome and specialization studies in Microbiology and Infectious Diseases at Sapienza University of Rome (Italy). She works as aggregate Professor in Department of Public Health and Infectious Diseases at Sapienza University of Rome. She is responsible for the Simple Operative Unit "Microbiological analyses in the immunocompromised hosts" and tutor of students and residents in the Department of Infectious Diseases. Her main research field is "The study of some microorganisms involved in human pathology focusing on MDR microorganisms such as KPC *Klebsiella, Acinetobacter baumannii and Helicobacter pylori as well as Candida* and on antibiotics activity". She has published 100 papers in reputed journals and has been serving as an Editorial Board Member of repute. She is an Editor of the book *Bacterial and Mycotic Infections in Immunocompromised Hosts: Microbiological and Clinical Aspects.* She is a reviewer of many important scientific international journals and research projects. She has attended many national and international conferences as speaker presenting relevant research topics.

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### Nurses as bullies and as victims of bullies

Gone are the days when the public perception of bullying was that it occurred mainly in residential neighborhoods and on school playgrounds, and that bullies were children whose parents had not taught them proper values and behaviors. This presentation will consider the serious problem of the bullying of nurses, sometimes by other nurses, who are employed in health care settings. Regardless of the job titles of the perpetrators, the bullying of nurses can have deleterious effects on a number of areas such as: Job performance, morale, productivity, turnover, and patient care, to name a few. This presentation will examine possible antecedents to such bullying behaviors as well as some of the interventions that have been undertaken to ameliorate those situations.

#### **Biography**

Carol Apt received her Ph.D. in Sociology from Northeastern University in Boston, Massachusetts (USA), her Masters in Sociology from Boston University in Boston, Massachusetts (USA), and her Bachelors in Sociology from Indiana University in Indianapolis, Indiana (USA). She also has a Certificate of French Studies from Ecole Lemania in Lausanne, Switzerland. As she was finishing her coursework at Northeastern University, she learned that she was the recipient of an internship in Applied Medical Sociology from the American Sociological Association. She chose to assume this honor at the University of Texas Medical School, Department of Psychiatry, in San Antonio, Texas, where she worked for 2 years. During this time she did HIV/AIDS research and wrote HIVAIDS education materials which she and her staff used with high-risk individuals incarcerated in the county jail in San Antonio. During this time she also taught at the University of Texas at San Antonio, and team taught a course Sociology of Genocide. She is also the host of a live, call-in radio program called "Talk to Me," which is broadcast on 90.3 FM-WSSB in South Carolina. The subject of her radio show is sexuality and relationships.

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# Keynote Forum Day 2

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## **Reinhard Buettner**

University Hospital Cologne, Germany

### **Experiences from the Network of Genomic Medicine (NGM)**

Traditionally, tumors are classified by histopathological criteria, i.e., based on their specific morphological appearances. To consequently, current therapeutic decisions in oncology are strongly influenced by histology rather than underlying molecular or genomic aberrations. The increase of information on molecular changes however, enabled by the Human Genome Project and the International Cancer Genome Consortium as well as the manifold advances in molecular biology and high-throughput sequencing techniques, inaugurated the integration of genomic information into disease classification. We have therefore introduced multiplex deep sequencing of informative gene sets into routine histopathological diagnostics and molecular pathology. This comprehensive approach integrating morphological and molecular information is currently changing cancer diagnostics in five categories: (1) Somatic genomic or epigenomic alterations acquired during cancerogenesis may be used for disease classification as we show this approach adding important information to conventional morphological classifications. (2) A significant portion of solid tumors depend on oncogenic driver lesions, which provide molecular targets for prediction of effective and selective therapies. (3) Genomic alterations in signal transduction cascades and gene expression pattern may be used as prognostic parameters predicting the need and extent of adjuvant therapy. (4) In the case of multiple syn- or metachronous tumors, genomic profiling assists allocation of metastases from tumors with unknown primary (CUP) and correct staging as multiple small primary tumors and systemic metastases are reliable being discriminated. (5) Finally, mutational profiling of tumor circulating tumor DNA may facilitate monitoring the response of tumors to therapy and development of secondary resistance.

Taken together, comprehensive molecular tumor pathology and oncology paves the way for a new rational and the basis of personalized genomic medicine. This requires state-of-the art tumor diagnostics and therapies in an interdisciplinary approach. Therefore, we will eview current technology and applications of NGS for molecular and predictive cancer diagnostics.

### Biography

Reinhard Büttner is Professor and Chairman of The Institute of Pathology at University Hospital Cologne, Köln, Germany, and the Co-Founder and Chief Scientific Officer of TARGOS Molecular Diagnostics. He completed his medical degree at the University of Mainz, Mainz, Germany, in 1985, before starting a residency at Rheinisch-Westfalische Technische Hochschule RWTH Aachen, Aachen, Germany. In 1987, he began post-doctoral work at the Gene Centre Munich and MD Anderson Cancer Centre, Houston, TX, USA (1987-1990). Returning to Germany in 1991, he took up a residency at the University of Regensburg, before becoming Professor and Chairman for Pathology at RWTH Aachen (1999-2001). After which, he worked as a Professor and Chairman of Pathology at the University of Bonn (2001-2011), before being appointed to his current position as Professor and Chairman of Pathology at the University of Cologne in 2011.

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Qihui Zhai

Mayo Clinic, USA

### Diagnosis of salivary gland tumors from morphology to molecular markers

Calivary glands tumors have numerous entities and each tumor type is of wide histologic spectrum. Clinical presentation is not Oparticularly helpful, with the usual presentation of a bump. The growth pattern of the tumor is a very critical histologic feature. If it is invasive and destructive, the tumor is very likely to be malignant. If it is well circumscribed/well demarcated, it is either a benign or a low grade tumor. Based on the presence of one cell type (luminal or non-luminal alone) or mixed luminal and non-luminal cell component (with an obvious extracellular matrix or not), we can classify most of the salivary gland tumors. Fine needle aspiration has been very useful in screening lesions with minimal trauma. However, previous FNA procedures can induce squamous metaplasia and tissue infarction, which sometimes misleads the pathologist. The metaplastic change also can mimic a low-grade mucoepidermoid carcinoma. On the other hand, a low-grade carcinoma such as cystic mucoepidermoid carcinoma is easily misdiagnosed a benign lesion, due to unimpressive bland cytological features. Immunohistochemistry studies are valuable when used along with histology; the main application is to demonstrate the cellular differentiation. Modern molecular tools such as FISH are important in separating those tumors with overlapping morphology. Translocations are found in adenoid cystic carcinoma (49%, MYB-NFIB), low and intermediate grade mucoepidermoid carcinoma (55%, CRTC1-MAML2), low-grade hyalinizing clear cell carcinoma (>80%, EWSR1-ATF1), and secretory carcinoma (>90%, ETV6-NTRK3). It is more stressful when we handle salivary gland tumors intraoperatively, because of the freezing artifacts and limited time, as well as the unavailability of ancillary tools. The combination of tumor demarcation, cell types, and cytological features can lead to correct diagnoses for most cases. For those rare and difficult cases, separating benign/low grade from high-grade tumors is usually sufficient to guide the immediate surgical procedure.

### Biography

Qihui Zhai is a Pathologist at Mayo Clinic. He received his Medical degree from Shandong Medical University and has been in practice for more than 20 years. He is one of 13 doctors at Mayo Clinic who has specialization in Pathology.

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## Jianhua Luo

University of Pittsburgh School of Medicine, USA

### Genomic drivers of human cancers: Diagnostics, prognostics and therapeutics

A ccurate prediction of clinical courses of human cancers remains elusive. In recent studies, we performed whole genome analysis on prostate and liver cancers. Our result showed that combination of genome copy number variance and novel fusion transcripts specific for cancer achieved high accuracy in predicting clinical outcomes of these cancers. Interestingly, some of these fusion genes are also present in a variety of human malignancies. Some of these fusion gene products trigger new pathways that are essential for carcinogenesis in multiple human cancers, and create novel functions that are not present in wild type gene counterparts. Treatment of cancers with drugs specific for fusion genes and their signaling pathways produced dramatic improvement of metastasis and survival rate of animals xenografted with cancers positive for these fusion genes. Our analyses suggest that targeting therapy for fusion genes holds promise as an effective treatment for human cancers.

#### **Biography**

Jianhua Luo has been studying Molecular Pathology related to human malignancies in the last 24 years. Currently, he is a Professor of Pathology and Director of High Throughput Genome Center at University of Pittsburgh. In the last 16 years, he has been largely focusing on "Genetic and molecular mechanism of human prostate cancer and hepatocellular carcinomas". In this period, his group has identified and characterized several genes that are related to prostate cancer and hepatocellular carcinomas. In this period, his group has identified and characterized several genes that are related to prostate cancer and hepatocellular carcinoma, including SAPC, myopodin, CSR1, GPx3, ITGA7, MCM7, MT1h and GPC3. He has characterized several signaling pathways that play critical role in prostate cancer development, including myopodin-ILK-MCM7 inhibitory signaling, myopodin-zyxin motility inhibition pathway, CSR1-CPSF3, CSR1-SF3A3 and CSR1-XIAP apoptotic pathways, MT1h-EHMT1 epigenomic signaling, ITGA7-HtrA2 tumor suppression pathway, GPx3-PIG3 cell death pathway, AR-MCM7 and MCM7-SF3B3 oncogenic pathways. He proposed prostate cancer field effect in 2002. He is one of the pioneers in utilizing high throughput gene expression and genome analyses to analyze field effects in prostate cancer. He is also the first in using methylation array and whole genome methylation sequencing to analyze prostate cancer. Recently, his group found that patterns of copy number variants of certain specific genome loci are predictive of prostate cancer clinical outcomes, regardless tissue origin. His discovery of several fusion transcripts and their association with aggressive prostate cancer has brought significant new insight into the field of prostate cancer of human malignancies.

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Sergey V Brodsky

Ohio State University, USA

### Anticoagulant related nephropathy: Lessons from patients and experimental animals

We have recently identified a new clinical syndrome in patients receiving warfarin for anticoagulation. This syndrome has been named warfarin-related nephropathy (WRN), and patients with chronic kidney disease (CKD) appear to be particularly susceptible. WRN is defined as an acute increase in INR to greater than 3.0, followed by evidence of acute kidney injury (AKI) within a week of the INR increase, defined as a sustained increase in serum creatinine of greater than or equal to 0.3 mg/dl. The AKI cannot be explained by any other factors, and the kidney biopsy demonstrates extensive glomerular hemorrhage with tubular obstruction by red blood cells. Beyond AKI, WRN is a significant risk factor for mortality within the first two months of diagnosis and it accelerates the progression of CKD. CKD is the most important risk factor for WRN and in CKD patients on warfarin who experience an increase in INR to >3.0, WRN is seen in 33-37% of the patients. Recent evidences suggest that WRN-like syndromes are not confined to anticoagulant-related nephropathy (ARN) to encompass the possibility that other anticoagulant drugs may put patients at risk. We developed an animal model to study ARN. 5/6 nephrectomy rats treated with warfarin or dabigatran showed increase in serum creatinine and morphology in the kidney similar to humans. Nephrologists and renal pathologists should be aware about this serious complication of anticoagulation therapy.

### **Biography**

Sergey V Brodsky has completed his MD and PhD in 1992 and 1995, respectively from North Ossetian Medical Academy in Russia. His research interests include renal pathology, renal physiology, vascular biology and angiogenesis. After finishing his Residency in Anatomic Pathology and a fellowship in Renal Pathology in 2009, he currently works as a Renal Pathologist at Ohio State University, USA. He has published more than 95 papers in peer-reviewed journals and book chapters.

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