13th International conference on

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Scientific Tracks & Abstracts

DAY 1

Sheng Xi Chen et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
DOI: 10.4172/2161-0681-C1-034

A Duo-toehold-mediated displacement amplification on DNA tetrahedron for RNA detection of dengue virus

Sheng Xi Chen, Ming Xuan Gao and **Sidney M. Hecht**
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Dengue is the most rapidly spreading endemic viral disease in the subtropical and tropical area of the world.This disease is threatening more than 2.5 billion people in more than 100 countries. Here, a novel duo-toehold-mediated displacement amplification strategy on DNA tetrahedron has been developed for sensitively detecting RNA of dengue virus. In this strategy, protector DNA was annealed to the tetrahedron to form a FRET-ON status at initial stage. When targets presented, the protector could be displaced by the target and switch to FRET-OFF. Meanwhile, the targets could be displaced again by the capture DNA and recycled in the process to amplify the signal. Simply by using the fluorescence spectrometer, the detection limit could be as low as 10 pM, which was more sensitive by 3 orders of magnitude than traditional non-amplified detecting methods.

Biography

Shengxi Chen has completed his PhD at the age of 27 years from Peking Union Medical Collegeand postdoctoral studies from University of Virginia. He is the Associate Professor Research in Center for BioEnergetics, Biodesign Institute, Arizona State University. He has published more than 30 papers in reputed journals.

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Notes:

Mark Podberezin, J Clin Exp Pathol 2017, 7:2 (Suppl)
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Diagnostic challenges in lung neuroendocrine tumors

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Neuroendocrine tumors (NET) of the lung constitute approximately 15% of all lung tumors, with small cell lung cancer (SCLC) accounting for 15% of invasive cancers. Many of those tumors have radiological and clinical presentation which is different from other pulmonary malignancies. In most cases, diagnosis could be established by core needle biopsy and, not uncommonly, SCLC is detected by endoscopic bronchial ultrasound fine needle aspiration (EBUS-FNA). Spectrum of lung NETs includes typical carcinoid (TC), atypical carcinoid (AC), SCLC and large cell neuroendocrine carcinoma (LCNEC). Morphological criteria, separating low grade from high grade NETs, include cellular atypia, mitotic rate, and presence or absence of necrosis. The question, which has been yet unanswered and which is addressed in the presentation, is whether the above NETs represent continuum from low to high grade tumors or they are biologically different. One of the major diagnostic challenges in pulmonary NETs is their grading on core needle biopsies (CNB). It has been shown that morphological features of NET, when diagnosed by CNB, could be significantly different from the ones on same tumor upon subsequent surgical resection. This could be partially due to marked crush and processing artifact which markedly affect evaluation of mitotic rate. Measurement of proliferative rate by immunohistochemical stain for Ki67 has been approved for grading of NET in the gastrointestinal tract, but is not universally accepted in pulmonary NET. However, it can be very helpful in evaluation of CNB with marked cautery and crush artifact. In addition, CNBs may not be representative of the entire lesion and can lead to diagnostic pitfalls which will be discussed in the presentation.

Biography

Mark Podberezin has completed his Medical School Degree (MD) and subsequent Clinical Hematology/Oncology training and PhD in Russia. Later, he did his Residency in Anatomic and Clinical Pathology at University of Illinois at Chicago and Hematopathology Fellowship at Texas Methodist Hospital in Houston. He is an Anatomic Pathologist (with special interest in Lung Pathology) and Hematopathologist at Royal University Hospital, University of Saskatchewan, Canada. He published 14 papers and presented at national, as well as international meetings.

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Notes:

Yaping Tian et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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Studies on miR-885-5p as a potential serum biomarker of HCC and its suppresses metastasis effects

Yaping Tian, Junhao Gui and **Zhuhong Zhang**
Chinese PLA General Hospital, China

Circulating miRNAs (microRNAs) are emerging as promising biomarkers for several pathological conditions, and the aim of this study was to investigate the feasibility of using serum miRNAs as biomarkers for liver pathologies. Real-time qPCR (quantitative PCR)-based TaqMan MicroRNA arrays were first employed to profile miRNAs in serum pools from patients with HCC (hepatocellular carcinoma) or LC (liver cirrhosis) and from healthy controls. Five miRNAs (i.e. miR-885-5p, miR-574-3p, miR-224, miR-215 and miR-146a) that were up-regulated in the HCC and LC serum pools were selected and further quantified using real-time qPCR in patients with HCC, LC, CHB (chronic hepatitis B) or GC (gastric cancer) and in normal controls. And then the miR-885-5p in HCC metastasis have been studied. The results demonstrated that the expression of miR-885-5p negatively correlated with the invasive and metastatic capabilities of human HCC tissue samples and cell lines. Overexpression of miR-885-5p decreased metastasis of HCC cells *in vitro* and *in vivo*. Inhibition of miR-885-5p improved proliferation of non-metastatic HCC cells. Furthermore, we disclosed that miR-885-5p targeted gene encoding β-catenin CTNNB1, leading to decreased activity of the Wnt/β-catenin signaling pathway. The present study indicates that miR-885-5p suppresses the metastasis of HCC and inhibits Wnt/β-catenin signaling pathway by its CTNNB1 target, which suggests that miR-885-5p to be a promising negative regulator of HCC progression and as a novel therapeutic agent to treat HCC.

Biography

Yaping Tian is a Professor of Department of Clinical Biochemistry, Chinese PLA General Hospital and Military Medical School. He is also a Professor of Nankai University, and Tsinghua University. He received his Master's degree in Medicine from Chinese PLA Postgraduate Medical School in 1989 and PhD from Academy of Military Medical Sciences in 1993. He had been trained as Postdoctoral Fellow for 2 years (1995-1997) in The Queen Elizabeth Hospital, Australia. He has been focusing on the study of specific serum proteomic profiles and genetic signatures in different diseases, especially on cancer and cardiovascular diseases. He also focused on the studies of antioxidants in herbal medicine and free radical biology. He has received more than 20 grants and published more than 300 scientific papers in peer-reviewed journals. He is on the Editorial Boards of several journals and the honor Chairman of the Clinical Biochemistry and Applied Molecular Biology Association, CSBMB.

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Notes:

Dipak P Ramji et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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Cytokines in atherosclerosis disease progression: Roles, mechanisms of actions and current therapeutic approaches

Dipak P Ramji, Thomas S Davies, Joe W E Moss, Jessica Williams, Ffion Harris, Alex Joseph, Faizah B Jaffar and Wijdan Al-Ahmadi
Cardiff University, UK

Atherosclerosis, the underlying cause of heart attack and stroke, is an inflammatory disorder of the vasculature regulated by both the innate and adaptive immune systems. Cytokines play a pivotal role in controlling the inflammatory response in atherosclerosis and regulate all the different stages in disease progression. Current approaches to target pro-inflammatory cytokines include neutralization using blocking antibodies or soluble decoy receptors and the use of specific inhibitors against key components of intracellular signaling pathways. In contrast, approaches for anti-atherogenic cytokines include their local delivery and the use of agents that augment their expression/actions. Numerous cytokines are expressed in atherosclerotic lesions and it is therefore essential that their actions in disease progression are fully understood to validate their therapeutic potential and to identify potentially new targets or approaches for therapeutic intervention. My laboratory has recently been investigating cytokine signaling in atherosclerosis, particularly in macrophages that play key roles in all stages of disease progression, using a combination of *in vitro* and *in vivo* approaches. Novel insights have been obtained on the actions of the cytokines interferon-gamma, transforming growth factor-beta, interleukin-33 and tumor necrosis factor-like protein 1A on key macrophage processes in atherosclerosis (e.g. foam cell formation, regulation of inflammation). For example, we have identified a key role for extracellular signal-regulated kinase: signal transducer and activator of transcription-1 serine 727 phosphorylation axis in the control of macrophage foam cell formation and the regulation of pro-inflammatory gene expression by interferon-gamma. The outcome of our studies on different cytokines will be presented in the context of current therapies and future developments in this field.

Biography

Dipak P Ramji received his BSc (Hons) degree (Biochemistry) and his PhD from University of Leeds. This was followed by Post-doctoral research at the EMBL (Heidelberg) and IRBM (Rome) with fellowships from the Royal Society and the EU. He joined Cardiff University in 1992 and is currently a Reader at Cardiff School of Biosciences. His research is focused on understanding how the immune and inflammatory responses regulate macrophage processes in atherosclerosis with the goal of attaining deeper mechanistic insight and identifying preventative/therapeutic agents. He has published over 80 peer-reviewed papers, reviews and book chapters (h-index=30; i10-index=57). He is an Editorial Board Member of 16 international journals.

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Notes:

Kunlun He et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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Mutation in *APOA5* gene associated with hypertriglyceridemia

Kunlun He, Wei Yan, Ruijun Yan, Chunlei Liu and **Yaping Tian**
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It is well accepted that the serum lipid level is modulated by genetic and environmental factors. Therefore, identification of the genetic variations involved in lipid metabolism could provide a clue to search for novel pathway in lipid regulation and thereby new therapeutic or preventive methods for coronary artery disease, and further improve the prognosis of heart failure and other cardiovascular disease. We extensively resequenced of our candidate genes and evaluated of rare variant accumulation to identify additional genetic variation responsible for increasing susceptibility to human hyperlipidemia. This study included 638 Chinese patients who were admitted to the Department of Cardiology, Chinese PLA General Hospital (Beijing, China) with chronic heart failure between January 2011 and January 2013. In total, 392 adult patients with hyperlipidemia and 246 population-based controls without hyperlipidemia were included in this study. In order to make the best possible to identify putatively damaging SNVs, five protein prediction algorithms (LRT score, MutationTaster, PolyPhen-2 HumDiv, PolyPhen-2HumVar and SIFT) were applied in non-synonymous SNVs. To explore the potential biological effects of the associated SNPs, we tested whether they were overlapped or correlated with the expression quantitative trait locus from four databases in peripheral blood cells. To mine the potential epistasis effects of the associated SNPs, we exhaustively searched all pairs among the variants. This high depth of resequencing study based on target genes repeatedly verified of two common mutation in *APOA5* gene associated with hypertriglyceridemia (11-116661392, 11-116662579). Besides, 3 rare variants in *APOA5* were also found to be related with the increase of triglycerides level. We detect the interaction effect across all pathways and discovered a stronger epistasis effect between gene *CNDP1* and *APOA5*, which both of them came from metabolic process and explained 7.55% genetic variance. We found common and rare variants associated with high triglyceride levels through the high depth of resequencing strategy. Much stronger associated signals are needed to excavate with large sample size and multicenter studies. Meanwhile, we also need studies of muti-omics to detect pathogenic mechanism of pathogenic variants.

Biography

Kunlun He has completed his Medical School degree from the Third Military Medical University, PhD from Chinese PLA Medical School, and Postdoctoral studies from College of Physicians and Surgeons of Columbia University. He is the Vice President of Chinese PLA General Hospital, and the Professor of Department of Cardiology. In recent years, he focuses on Translational Medicine of Cardiovascular disease. He has published more than 158 peer reviewed papers, achieved the first class awards of Beijing Science and Technology, and also he has been serving as an Editorial Board Member of three medical journals.

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Malgorzata Jaremko et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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A framework for translating advances in molecular genetic technologies into clinical laboratory practice

Malgorzata Jaremko and **Alexander Bisignano**
Phosphorus Diagnostics, USA

Advances of the molecular diagnostic testing platforms, including development and implementation of NGS based genetic testing contribute to the improvement of disease prediction, diagnosis, and treatment. However, the future of genomic medicine relies on the capability of molecular genetics laboratories to develop and validate evidence-based and cost-effective laboratory tests. These laboratories face many challenges including establishing clinical utility, validating analytical performance of laboratory developed tests, and managing costs of platform development and subsequent consumables. Along with the molecular and instrumentation challenges, laboratories are faced with a myriad of software options (e.g., Galaxy, Amazon, GATK, BaseSpace, and Clarity) when establishing a reliable bioinformatics pipeline and LIS system. Finally, there is a lack of consensus and consistency in the quality standards across the industry (e.g., read depth, variant curation, and clinical validation structure). In this study, we present a framework for the consistent development of accurate, high-quality, NGS diagnostic tests. Our process is broken into stages from gene selection through clinical validation and implementation. Based on the experience in our own CLIA-laboratory, we present lessons learned in the development of NGS targeted panels for sequencing and CNV analysis for various indications including infertility, hereditary cancers, arrhythmias, cardiomyopathies and lipidemias.

Biography

Malgorzata Jaremko has completed her PhD in Pharmacogenomics from Wroclaw Medical University and Postdoctoral Clinical Fellowship from Mount Sinai School of Medicine, NY. She is board certified by American Board of Medical Genetics and Genomics in Clinical Molecular and Clinical Biochemical Genetics; and she is Fellow of American College of Medical Genetics and Genomics, as well as National Academy of Biochemistry. She has extensive experience in directing clinical molecular laboratories, and currently serves as the Senior Director, Clinical Laboratory & Molecular Diagnostics, and CLIA-Director of Phosphorus Diagnostics genetic testing laboratory.

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Melanie Yong, J Clin Exp Pathol 2017, 7:2 (Suppl)
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The integrated 152 solid tumor panel for early-line and advanced stage treatment decisions in solid tumors

Melanie Yong
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Comprehensive genetic profiling of tumors using next generation sequencing (NGS) is gaining acceptance for guiding treatment decisions in cancer care. We designed a cancer profiling test that integrates results from NGS with more traditional results obtained by immunohistochemistry (IHC) of solid tumor tissues. Relevant regions of genes known to be implicated in solid tumors are targeted for deep sequencing. The tight concordance between some somatic mutations and the standard-of-care (SOC) therapeutics administered in clinical practice makes identification of such mutations in a specific tumor invaluable in guiding personalized and rational treatment of the patient. The SOC report is provided in a short turnaround time for four tumors, namely lung, breast, colon and melanoma, followed by a full report that includes drug candidates available through clinical trials. For all other tumor types, a full report is provided. Our Integrated 152 solid tumor panel not only detects single nucleotide polymorphisms (SNPs), but will identify copy number variations (CNVs) and some translocations in 152 cancer-related genes. We describe the standardization, validation, and clinical utility of the Integrated 152 Solid tumor test on approximately 250 solid tumor formalin-fixed paraffin-embedded (FFPE) disease samples and control cell-line samples. These studies showed high reproducibility and accuracy (~99%). When therapeutics in clinical trials was included, clinically relevant recommendations increased to 95% for patients in advanced stages of cancer. We present data to demonstrate how the Integrated 152 Solid Tumor Test may be used in clinical practices.

Biography

Melanie Yong is the Senior Manager at Integrated Molecular Diagnostics Pathology, Inc. (IMD Path). She earned her Bachelor's of Science in Biomedical Sciences and Microbiology from Colorado State University and certified by American Society for Clinical Pathology (ASCP) Board of Certification in Molecular Biology, MB (ASCP)CM. She is committed to the highest standard of excellence and enjoys the challenges that come along with new science and technology in the advancing Biotechnology and clinical diagnostics field. Her current research focuses in cancer genomics.

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Sponsored Workshop

Day 1

Frederick Husher et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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Frederick Husher & Jee Shum

HS Technology LLC, USA

PRS – co-resident objective measure of IHC stain performance for process QC and diagnostic aid

The Process Record Slide (PRS) records the ImmunoHistoChemical (IHC) stain processing experience of a co-resident patient tissue section using arrays of stain reagent detection targets. Both experience all the IHC processing from tissue capture to the application of the cover slip: tissue capture, drying, deparaffination, antigen retrieval, primary antibody, and secondary amplification processing. Because the PRS targets are comprised of known reactivity concentrations to the stain reagents, an objective measure that is unique to that slide now exists remaining forever co-resident with the tissue section. The result is a captured efficacy record of the antigen recovery, stain reagents, and the processing technology. The PRS targets can be used with digital imaging to quantify the IHC processing upon the tissue section using the reference scales developed from the targets. The reference scales can be used for objective determination of antigen density in the tissue and QC reporting of the process. Additionally, utilizing the reference scales, the tissue section image presentation can be normalized to a preferred basis upon which optimal diagnostic determination can be achieved. Tele-diagnostics and second opinion are also possible since the unique processing experience is recorded. Others have attempted to produce control slides but have all failed in meeting the constraints of mass production at an affordable price. Thus, only with the development of a new slide coating that meets the covalent binding needs of target & tissue, target printing technology, and production automation, can the goals be satisfied. PRS technologies satisfies these goals.

Biography

Frederick Husher and Jee Shum have pursed and refined the development of the PRS for more than a decade and they have successfully resolved many technology challenges including covalent adhesive slide coatings supporting both tissue and proteins, non-staining label paint, bio-target printing, and bio-target fabrication. A joint venture with the Hong Kong Productivity Council, to co-develop the production technology, will bring the PRS into commercial reality to benefit global health.

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Scientific Tracks & Abstracts

DAY 2

Mark Podberezin, J Clin Exp Pathol 2017, 7:2 (Suppl)
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Multiple myeloma with plasmablastic morphology and central nervous system (CNS) involvement

Mark Podberezin
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We describe an unusual case of multiple myeloma (MM) with plasmablastic morphology in a young man who, after chemotherapy and autologous stem cell transplant, developed disease recurrence with isolated CNS involvement. Plasmablastic type is characterized by immature cells with round nuclei, prominent central nucleoli, and amount of cytoplasm much smaller than that in mature plasma cell. According to different studies, plasmablastic MM may account for 15-30% of MM cases and tend to be associated with adverse prognosis. It has been shown to correlate with high proliferation rate, extensive bone marrow infiltration, but not with high risk chromosomal aberrations. Despite the fact that plasmablastic morphology is not uncommon in MM patients, CNS involvement in this disease is very rare. Overall, extramedullary involvement is found in 7% of patients with MM upon initial diagnosis, with CNS involvement occurring in less than 1% of patients. Based on Mayo Clinic study of 4060 MM patients, only 0.7% of all patients had CNS disease. MRI studies demonstrate that predominant pattern of CNS myelomatous disease is leptomeningeal involvement, with intraparenchymal tumor-like lesions being much less common. In some patients, dural involvement and/or direct extension of MM into CNS were described. In 82% of patients with CNS myelomatosis, neoplastic plasma cells were found in CSF, and in those without CSF involvement, the diagnosis was made by MRI which detected either leptomeningeal or intraparenchymal involvement. It is worth to mention that CNS involvement in MM tends to occur in younger patients, without evidence of advanced disease, sometime with isolated CNS involvement. In many of these patients, complete remission can be achieved with the use of systemic, rather than intrathecal, chemotherapy, particularly with addition of novel agents such as Bortezomib (proteasome inhibitor) and Lenalidomide (immunomodulatory agent). Diagnostic approach, as well as management of patients with CNS involvement by MM, will be discussed.

Biography

Mark Podberezin completed his Medical School (MD) Degree in Russia where he practiced Clinical Hematology before moving to USA. He performed his PhD project in Immunohematology in United Kingdom. After moving to USA, he completed his Anatomic and Clinical Pathology Residency, as well as Surgical Pathology Fellowship training at University of Illinois at Chicago/Cook County Hospital and later completed Hematopathology Fellowship at Texas Methodist Hospital in Houston. Currently, he is Anatomic Pathologist/Hematopathologist and Assistant Clinical Professor at University of Saskatchewan, Canada.

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Notes:

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Nutraceuticals as promising agents in the prevention and treatment of atherosclerosis

Dipak P Ramji, Thomas S Davies, Hayley Gallagher, Joe W E Moss, Jessica Williams, Wijdan Al-Ahmadi and **Victoria O'Morain**
Cardiff University, UK

Atherosclerosis, an inflammatory disorder of medium and large arteries and the underlying cause of myocardial infarction and cerebrovascular accident, is responsible for more deaths worldwide than any other disease. Pharmaceutical intervention together with lifestyle changes have recently resulted in a slight reduction in morbidity and mortality from atherosclerosis and its complications, at least in the western world. However, this is expected to change in the future, because of global increase in risk factors such as obesity and diabetes. Current pharmaceutical therapies against atherosclerosis such as statins are not fully effective and associated with several side effects together with patient-dependent efficacy. Unfortunately, many pharmaceutical leads against established targets have proved disappointing at the clinical level (e.g. inhibitors against cholesterol ester transfer protein). It is therefore essential that further research is carried out into alternative therapies for the prevention and/or treatment of atherosclerosis. Nutraceuticals have recently received substantial interest for the prevention/treatment of atherosclerosis. However, more in-depth understanding is required on the molecular mechanisms underlying the actions of nutraceuticals together with large clinical trials testing their efficacy. We have recently initiated studies on the effects of many nutraceuticals, including certain omega-6-fatty acids, polyphenols and flavanols, on several key monocyte/macrophage processes associated with atherosclerosis *in vitro* (e.g. monocytic migration, macrophage polarization, foam cell formation, activation of inflammasome and production of reactive oxygen species) and various risk factors *in vivo*. These will be presented in the context of current therapies and those that are being developed.

Biography

Dipak P Ramji received his BSc (Hons) degree (Biochemistry) and his PhD from University of Leeds. This was followed by Post-doctoral research at the EMBL (Heidelberg) and IRBM (Rome) with fellowships from the Royal Society and the EU. He joined Cardiff University in 1992 and is currently a Reader at Cardiff School of Biosciences. His research is focused on understanding how the immune and inflammatory responses regulate macrophage processes in atherosclerosis with the goal of attaining deeper mechanistic insight and identifying preventative/therapeutic agents. He has published over 80 peer-reviewed papers, reviews and book chapters (h-index=30; i10-index =57). He is an Editorial Board Member of 16 international journals.

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Notes:

Hicran Şenli et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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Investigation of relationship between -1195 A>G polymorphism of *COX-2* gene and mRNA levels of *COX-2* gene in peripheral blood monocyte in colorectal cancer patients

Hicran Şenli, Leyla Bahar, Nazan Eras, Tahsin Çolak, Mehmet Ozgur Turkmenoglu, Seval Kul and Etem Akbas
Mersin University, Turkey

Colorectal cancer (CRC) arises from the colorectal epithelium as a result of the accumulation of genetic alterations in defined oncogenes and tumor suppressor genes. The molecular changes occurring during the development of the tumor must be investigated in order to understand the carcinogenesis. The cyclooxygenase (COX) isoenzymes, COX-1 and COX-2, catalyze the formation of prostaglandins, thromboxane, and levuloglandins. *COX-2* is induced by inflammatory and mitogenic stimulants and prevails on tumor carcinogenesis by increasing the prostaglandin synthesis in inflammatory and neoplastic tissues. The aim of this study was to investigate the association the *COX-2* gene -1195 A>G polymorphism and CRC risk. We also investigated the relationship between the *COX-2* gene mRNA levels in peripheral blood monocytes and -1195 A>G polymorphism in CRC. Ninety individuals with CRC and 106 healthy individuals are included in our study. The genotypes are determined by using PCR-RFLP. RNA of individuals with CRC is isolated and RT-PCR is applied. Genotype distribution and allelic frequencies for -1195 A>G polymorphism of *COX-2* gene weren't significantly different between patients and controls. *COX-2* gene mRNA levels and genotype distributions of this polymorphism has no difference between CRC patients and controls. While one of the other factors of developing CRC; the advanced age and male gender increases the risk of developing CRC, BMI, smoking and alcohol intake have no effect on risk of developing CRC. Our study is the first study to investigate the relation between -1195 A>G polymorphism and mRNA levels of *COX-2* gene in CRC in Turkish population.

Biography

Leyla Bahar, after graduating from Cukurova University Faculty of Medicine, worked as a Medical Practitioner in Mersin, until 2002. In the Department of Histology-Embryology, she completed PhD in Mersin University Faculty of Medicine in 2008. She has published more than 20 papers and announcement in journals and has been serving as a Consultant Editor and Editorial Board Member of reputable journals. She still continues to work as a Scientist and an Assistant Professor at Mersin University, who is working on many issues and as peer-reviewer in journals.

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Notes:

Melek Meydanogulları et al., J Clin Exp Pathol 2017, 7:2 (Suppl)
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Investigation for polymorphisms of *caspase 3* and *caspase 9* gene and enzyme levels in leukemia patients

Melek Meydanogulları, Nazan Eras, Naci Tiftik, Anil Tombak and Etem Akbas
Mersin University, Turkey

For understanding of leukemia and its treatment with early diagnosis, the molecular changes that occur during the development of leukemia should be investigated. Apoptosis is central to the development and homeostasis of the hematopoietic system. Previous studies have reported that leukemia cells invariably have abnormalities in one or more apoptotic pathways. The current study investigated the relationship between polymorphisms of *caspase 3* G>T rs4647601 and *caspase 9* A>G rs4645978 and leukemia. Besides that, we aimed to determine *caspase 3* and *caspase 9* enzyme levels possible effects on the risk of developing leukemia. The case group consisted of 100 patients (mean age: 56±03) who had been newly diagnosed with leukemia at the Department of Hematology, Mersin University Faculty of Medicine, Turkey. The control group comprised of 100 healthy properly age and sex matched individuals (mean age: 54±15) with a no history of leukemia. The genotypes were detected by using Real-Time PCR. We measured enzyme levels of *caspase 3* and 9 in serum, which were obtained from blood samples. No significant association was observed between *caspase 3* G>T rs4647601 and *caspase 9* A>G rs4645978 polymorphisms and leukemia. We found that median levels of *caspase 3* and 9 were higher in leukemias than in normal blood cells (P<0.001). This is the first study reporting the detailed distribution of alleles and genotypes of *caspase 3* and *caspase 9* in leukemia patients in Turkish population. Taken together, we conclude that *caspase 3* and *caspase 9* levels may be useful for the early diagnosis of leukemia.

Biography

Nazan Eras, after graduation from Dicle University Faculty of Medicine, worked as a General Practitioner. She completed her MSc in 2006 and PhD in 2012 at Mersin University Faculty of Medicine, Department of Molecular Biology and Genetics. She has been serving as Peer-reviewer in journals. She still continues to work as a Scientist and an Assistant Professor at Mersin University Medical Faculty, Department of Medical Genetics. Her research interests include Clinical Cancer Genetics, Human Molecular Genetics and Oxidative Stress.

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Notes:

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Workshop
Day 2

Bevan Tandon, J Clin Exp Pathol 2017, 7:2 (Suppl)
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Bevan Tandon

Molecular Pathology Laboratory Network, Inc., USA

Contemporary genomic studies in hematologic pathology: Utility of next generation sequencing in clinical evaluation of myeloid and lymphoid malignancies

Next generation sequencing (NGS) methodologies are emerging as an extremely valuable adjunct in the clinical diagnostic evaluation of hematologic cancers. Simultaneous assessment for prognostic or therapeutically predictive mutations across numerous disease relevant genes can be easily accommodated by clinical targeted NGS panels, facilitating significant reductions in labor, cost, and turnaround time for clinical reporting. Multiplex targeted NGS panels also eliminate reliance upon cascaded mutation testing algorithms often found to be highly complex and cumbersome for ordering physicians. Thus, extended mutational profiling using NGS may show significant utility in the evaluation of acute myeloid leukemias and myeloproliferative neoplasms. Targeted NGS gene panels have also been reported to show potential, emerging significance in evaluation of myelodysplastic syndromes, one of the most common clinical indications for bone marrow biopsy. In the setting of acute leukemias and mantle cell lymphoma, minimal residual disease (MRD) testing by NGS has also been reported to show significant improvements in sensitivity and specificity compared to the standard reference methodologies including flow cytometry and PCR. Lastly, Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma (CLL/SLL) is the most common leukemia diagnosed among adults in western countries and is associated with heterogeneous clinical outcomes. Somatic hyper mutation status of the IGH gene is one of the most important prognostic biomarkers for risk stratification and guidance of therapy in this setting, and NGS confers significant practical and technical advantages over the current gold standard Sanger Sequencing based approach.

Biography

Bevan Tandon, MD is board certified by the American Board of Pathology in both Hematologic Pathology and Molecular Genetic Pathology. His Hematopathology training was completed at the University of Pittsburgh under the guidance of WHO lead Author, Steven Swerdlow. His Molecular Pathology training at Washington University in St. Louis was focused on next generation sequencing for clinical testing in Oncology. He has multiple publications in the peer reviewed literature including the International Journal of Laboratory Hematology and Modern Pathology. He currently serves as the Director of Clinical Molecular Diagnostics at Molecular Pathology Laboratory Network, Inc., USA.

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