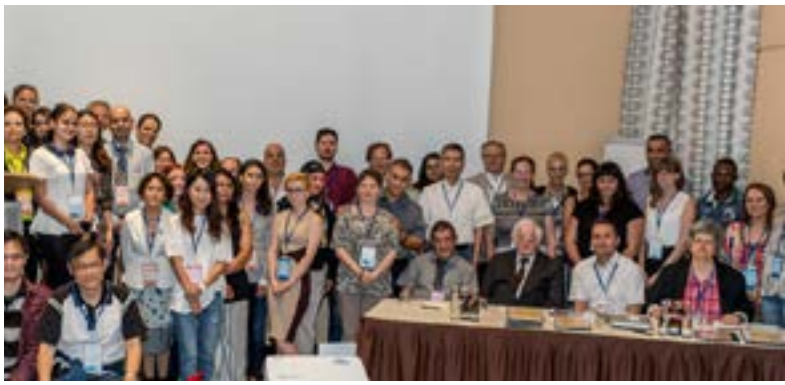

International Conference on

Gastrointestinal Cancer and Therapeutics &

4th World Congress on

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26th Annual Congress on

Cancer Science and Targeted Therapies

October 29-30, 2018 | San Francisco, USA

Keynote Forum

Day 1

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October 29-30, 2018 | San Francisco, USA

Michael W Retsky

Harvard TH Chan School of Public Health, USA

Solution proposed to a 2000-year-old problem in oncology: Recent developments

A bimodal pattern of hazard of relapse among early-stage breast cancer patients has been identified in multiple databases from US, Europe and Asia. We are studying these data to determine if this can lead to new ideas on how to prevent relapse in breast cancer. Using computer simulation and access to a very high-quality database from Milan for patients treated with mastectomy only, we proposed that relapses within 3 years of surgery are stimulated somehow by the surgical procedure. Most relapses in breast cancer are in this early category. Retrospective data from a Brussels anesthesiology group suggests a plausible mechanism. Use of ketorolac, a common NSAID analgesic used before surgery was associated with far superior disease-free survival. The expected prominent early relapse events in months 9-18 are reduced 5-fold. Transient systemic inflammation accompanying surgery (identified by IL-6 in serum) could facilitate angiogenesis of dormant micrometastases, proliferation of dormant single cells, and seeding of circulating cancer stem cells (perhaps in part released from bone marrow) resulting in early relapse and could have been effectively blocked by the perioperative anti-inflammatory agent. If this observation holds up to further scrutiny, it could mean that the simple use of this safe, inexpensive and effective anti-inflammatory agent at and perhaps after surgery might eliminate early relapses. We suggest this would be most effective for triple negative breast cancer and be especially valuable in low and middle-income countries. Similar bimodal patterns have been identified in other cancers suggesting a general effect. Based on their writings, Galen and Celsus knew of this 2000 years ago.

Biography

Michael W Retsky (PhD in Physics from University of Chicago) made a career change to cancer research thirty years ago. He is Research Associate at Harvard TH Chan School of Public Health and Honorary Reader at University College London. He was on Judah Folkman's staff at Harvard Medical School for 12 years. Retsky is Editor of a Springer-Nature book on the breast cancer project published July 2017. After diagnosis of stage IIIc colon cancer in 1994, he was the first person to use what is now called metronomic chemotherapy. He is a founder and for 10 years was on the Board of Directors of the Colon Cancer Alliance. He has published more than 70 papers in physics and cancer.

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October 29-30, 2018 | San Francisco, USA

Andrey Sorokin

Medical College of Wisconsin, USA

p66Shc regulates vascular dysfunction and renal damage in diabetic nephropathy

Patients with poorly-controlled diabetes mellitus sustain damage to the macro and microvasculature that is responsible for much of the morbidity and mortality associated with the disease. In the kidney, dysregulation of glomerular blood flow has been implicated as one factor in the pathogenesis of diabetic glomerulosclerosis. Increased expression of adaptor protein p66Shc has been associated with progression of diabetic nephropathy. Afferent arteriolar dilation and glomerular hyperfiltration in diabetes are due to increased KATP channel availability and activity. Hyperglycemia was induced in Dahl SS (SS) rats in a model of type 1 diabetes via injection of streptozotocin (STZ). Albuminuria and glomerular injury were evaluated in SS and genetically modified SS lacking either p66Shc (p66ShcKO) or expressing p66Shc mutant (p66Shc-S36A). Afferent arteriolar diameter responses during STZ-induced hyperfiltration were determined using the juxtamedullary nephron technique to assess the role of p66Shc in KATP activity. Albuminuria and glomerular injury were mitigated in p66ShcKO and p66Shc-S36A rats with STZ-induced diabetes. SS rats with STZ-induced diabetes had a significant increase in the afferent arteriolar diameter, whereas p66ShcKO and p66Shc-S36A rats did not. STZ SS rats, but not STZ p66ShcKO or p66Shc-S36A rats had an increased vasodilator response to KATP channel activator pinacidil. Likewise, KATP inhibitor glibenclamide resulted in a greater decrease in afferent arteriolar diameter in STZ SS rats compared to STZ-treated SS p66ShcKO and p66Shc-S36A rats. Taken together, these results indicate that deletion of the adaptor protein p66Shc decreases afferent arteriolar KATP channel activity and decreases renal damage in diabetic SS rats.

Biography

Andrey Sorokin graduated from the St Petersburg State University and received his PhD from the Institute of Cytology Academy of Sciences of Russia in 1981. He is a Head of the Laboratory at the Medical College of Wisconsin, where he is holding the position of Full Professor with secondary appointments at Department of Physiology and Department of Microbiology & Immunology. He has published more than 100 papers in reputed journals and serving as an editorial board member of a number of journals including Frontiers in Renal and Epithelial Physiology.

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Octavian Bucur

BIDMC, USA

Physical tissue expansion for nanoscale investigation of clinical specimens

Objective: In pathology, examination of cellular structures and molecular composition using diffraction-limited microscopy is key to diagnosis. Recently, a new approach, Expansion Microscopy, was developed, enabling physical magnification and high resolution imaging of cell lines and mouse brain sections with conventional optical microscopes, by embedding them in a dense swellable polymer and adding water to swell the polymer after the enzymatic digestion of the proteins (Chen F et al., Science, 2015). The purpose of our study is to develop a pathology-optimized physical tissue expansion method for nanometer imaging and investigation of clinical tissue samples and to analyze its utility in diagnostic pathology and research.

Methods: We developed a pathology optimized physical tissue expansion methods called Expansion Pathology (ExPath), which uses clinically optimized chemistry, labeling and imaging methodologies to expand and visualize both human FFPE and frozen clinical samples, including previously stained/unstained, mounted/unmounted and whole tissue slide/tissue microarrays sections, of a wide variety of fixed human tissue types and pathologies.

Results: This ExPath protocol enabled expansion of human normal and cancer tissues (including gastrointestinal malignancies) ~4.5x in linear dimension and ~100x in volume, with a post-expansion measurement error of 3-7%. Physical tissue expansion pushes the optical microscopes beyond their limits (currently 250nm in resolution), by enabling for the first time ~70nm resolution imaging of diverse biomolecules in intact tissue with an optical microscope. With ExPath, certain lesions and pathologies previously diagnosed with an electron microscopy (EM) can now be diagnosed with a conventional optical microscope after physical tissue expansion, an inexpensive, faster and reliable strategy. It also enables high-fidelity computational discrimination between early neoplastic lesions that to date have challenged the human judgment.

Conclusion: ExPath offers new approaches for assessing pathologically important features in human tissue. It may eliminate the need for EM in the diagnosis of certain diseases for which EM is required for diagnosis and it can improve the computational discrimination between pathological lesions that are hard to distinguish with existing techniques. ExPath may enable routine use of nanoscale imaging in molecular pathology and research (2017, Nature Biotechnology).

Biography

Octavian Bucur, MD, PhD is Instructor in the Department of Pathology at the Harvard Medical School and Beth Israel Deaconess Medical Center, in Boston, MA, focusing on the development and application of new experimental and computational technologies with significant impact in molecular, diagnostic pathology and personalized medicine. He is also a member of the Ludwig Cancer Center at Harvard and Broad Institute of MIT and Harvard. In collaboration with Dr Edward Boyden's laboratory at MIT, he has developed a pathology-optimized physical tissue expansion method called Expansion Pathology, that enables ~100 times expansion in volume of any type of clinical specimen and visualization of 70-80nm structures with conventional optical microscopes (currently limited to ~250nm resolution). Expansion Pathology has the potential of replacing electron microscopy in diagnosis and investigation of certain pathologies and nanometer structures (Nature Biotechnology, August 2017, 3 patents filed).

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Mohamed El Far

Mansoura University, Egypt

Breakthrough in photodynamic therapy of tumors: Our 40 years experience from bench to clinical applications

We will present an overview of our long-term team(s) experiences in photodynamic therapy (PDT) of tumors by Lasers from drug discovery to clinical applications. Main topics: (1) Our search for ideal tumor localizers and photosensitizers. (2) Modification and synthesis of newly developed photosensitizers to enhance the efficacy of PDT. (3) Novel approaches for the removal of skin photo-toxicity after PDT treatment. (4) Our newly developed technique for diagnosis and treatment of bladder cancer using 5-ALA as well as the mechanism of its biodistribution and action. (5) The potential use of other light sources in PDT. (6) PDT- clinical applications. (7) Where do we go from here?

Biography

Mohamed El Far worked in the biochemistry field for 40 years, published over 90 peer-reviewed papers. He received Fulbright & British council fellowships several times, German DAAD grant to establish PDT Program at Munich, received US-AID grant to establish PDT unit in Egypt. He is serving on the editorial boards/Editor to four international journals. Acts as UNESCO expert in science and technology. He served as visiting professor to the University of California, Utah laser center, Mayo clinic, and Cardiff and Swansea Universities, UK several years. Member of International Photodynamic Association and Royal Society of Chemistry, UK. Selected among top 100 scientists 2012.

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Notes:

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Diana Anderson

University of Bradford, UK

Comparison of aspirin and ibuprofen bulk and nanoforms in peripheral lymphocytes from breast cancer patients and healthy individuals

Epidemiological studies have suggested that regular intake of some non-steroidal anti-inflammatory drugs (NSAIDs) have a preventative effect against several types of tumors including breast cancer in humans. This present study aims to investigate the effect of both ibuprofen and aspirin on DNA damage using lymphocytes obtained from breast cancer patients and comparing the result with lymphocytes from healthy females as a control. Lymphocytes are useful surrogates for cancer cells. Nanoparticles (NPs) and bulk sizes were used in the Comet and micronucleus assays. 250ng/ml of ibuprofen (NPs and bulk) and 500ng/ml of aspirin were used as non-toxic doses to treat the lymphocytes. Aspirin, both bulk and nano sizes, showed a significant reduction in DNA damage in the Comet and micronucleus assays. However, the effect of aspirin nano ($P \leq 0.01$) was more significant compared to aspirin bulk ($P \leq 0.05$). Ibuprofen, in contrast, showed a significant reduction in micronucleus (MNi) frequency in the micronucleus assay with the nano form ($P \leq 0.001$) being more significant than the bulk form ($P \leq 0.01$), whilst its preventative effect with the Comet assay was insignificant. These observations suggest that NPs have better penetration through the nuclear membrane due to their smaller sizes compared to their bulk size. Aspirin was more effective than ibuprofen in the reduction of DNA damage and MNi formation in the Comet and micronucleus assays. NPs were more effective than bulk sizes. The results are consistent with the view that NSAIDs, particularly aspirin and ibuprofen, could have a promising role in cancer treatment including breast cancer.

Biography

Diana Anderson holds the Established Chair in Biomedical Sciences at the University of Bradford. She obtained her first degree in the University of Wales and second degrees in the Faculty of Medicine, University of Manchester. She has 460+ peer-reviewed papers, 9 books, has successfully supervised 30 PhDs, and been a member of editorial boards of 10 international journals. She has been or is Editor in Chief of a book Series on toxicology for J Wiley and sons and the Royal Society of Chemistry respectively. She gives key note addresses at various international meetings. She is a consultant for many international organisations, such as the WHO, NATO, TWAS, UNIDO and the OECD. Her h index = 59.

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Pegah Varamini

University of Sydney, Australia

Development of a peptide-functionalised drug delivery system for targeted therapy of advanced colon cancer

Colorectal cancer is the third most frequent cancer in the Western world {Jemal, 2005 #3160}. In patients diagnosed at an early stage of colorectal cancer, surgical excision followed by adjuvant radiation or chemotherapy leads to a high degree of response and improves the survival rates. However, therapeutic options for advanced or disseminated cases are limited, and the responses to treatment are generally temporary. Thus there is an urgent need for the development of new, more efficient and targeted therapeutic modalities. High levels of the luteinizing hormone-releasing hormone (LHRH) receptors have been demonstrated in sex steroid-dependent tumors such as breast and prostate cancers, and also in malignancies that are not directly influenced by the pituitary-gonadal axis like colon cancer. We have taken advantage of this differential receptor expression by attaching a new derivative of the LHRH peptide to the outer surface of novel polymer nanoparticles. These nanoparticles are loaded with curcumin as the model drug, a non-toxic plant extract that has recently attracted much attention in medicine due to its remarkable therapeutical actions. It is called the "next generation multi-purpose drug" and is the active constituent of the Indian spice turmeric. However, it suffers from a very poor metabolic stability and bioavailability due to low water solubility. In this study, we have demonstrated that our advanced formulation strategy has overcome many of the hurdles associated with poorly soluble drug molecules like curcumin. This drug delivery system has shown promising potentials to be effectively used as a medication and also target anticancer agents specifically to the colon cancer cells.

Biography

Pegah Varamini is a lecturer and group leader in Cancer Theme within the Faculty of Pharmacy. She is the leader of Cancer Targeting-Drug Delivery Group. She was awarded the prestigious National Breast Cancer Foundation (NBCF) fellowship in Jan 2016. She completed her PhD degree in Medicinal Chemistry and Pharmacology in December 2012 (UQ, Australia). She also has a professional Doctorate degree in Pharmacy (PharmD). She won Dean's Award for Research Higher Degree Excellence in 2013. Her work was selected by the Australian Academy of Science in August 2016, resulting in her personal presentation at the inaugural Falling Walls Lab in Canberra (a gathering of 25 selected Australian and New Zealand researchers, entrepreneurs, engineers and innovators). She has been the Collaboration Award Finalist at Sydney University in 2017.

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Madhusudhanan Jegadeesan

Velammal Medical College, India

Surgery for acute pancreatitis: Principles and practice

Acute pancreatitis is the most common pancreatic pathology encountered in India. Majority of them are caused by ethanol consumption while biliary etiology accounts for the rest. Young alcoholic males and middle-aged obese females are usually affected. Another cause, peculiar to the Tropics, is the chronic calcific pancreatitis with acute exacerbations. This population usually consists of boys and girls in their teens. In most situations, acute pancreatitis is a mild and self-limiting disorder. Initial care depends on aggressive fluid management and pain control. This usually happens in the first 3-4 days of the illness where the patient will be managed in a community health setting. However, in a tertiary referral center, the majority of acute pancreatitis patients have a complicated course which necessitates surgical management. Radiologically, patients with severe acute pancreatitis have fluid collections around the pancreas along with variable degrees of necrosis of the gland. Understanding the nature of the fluid collection and the extent of necrosis in conjunction with the clinical condition of the patient helps in accurate management. As a teaching hospital, our surgical unit treats more than 300 patients with acute pancreatitis every year. This talk will focus on the scientific principles that guide the surgical management of acute pancreatitis patients.

Biography

Madhusudhanan Jegadeesan had completed his training in General Surgery in 2008 and passed out with Dr TMA Pai Gold Medal from one of the most reputed Institutions in India. Later, he specialized in Gastrointestinal Surgery from Madras Medical College, Chennai, India which is the oldest medical college in Asia having started in 1835. He had his advanced training in Hepatopancreaticobiliary surgery and Liver Transplantation from Apollo Hospitals, New Delhi which performs more than 300 liver transplants every year. He has numerous publications to his credit and has been invited to deliver talks in South Korea and Japan. He had also been invited to demonstrate major esophageal, liver and pancreatic surgeries in medical colleges and Cancer Institutes in various parts of India. He presently leads the Hepatobiliary and Liver Transplant Unit at Velammal Medical College, Madurai, India.

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Edward Lichten

Wayne State University School of Medicine, USA

The paradigm shift in the diagnosis and medical treatment of inflammatory bowel disease

One of the most destructive benign diseases of young adults is inflammatory bowel disease (IBD). This includes Crohn's disease, ulcerative colitis, and microscopic colitis. As a result of medication treatment failures, upwards to two-hundred thousand young men and women in North America will have major bowel resection annually. More than half of these 1.6 million IBD sufferers will experience a premature death compared with the general population; this includes an increased risk of colorectal cancer. The incidence of the disease is increasing: a 10 percent increase has been noted in the youngest pediatric population over the last 20 years. The problems to date in understanding and treating IBD are multifactorial: no one has identified the cause, no specific biomarkers are recognized, there are no alternatives to overtly toxic medications, and no proven alternatives presently exist to the inevitable surgical resection and disease recurrence. This review of the medical literature is admixed with the initial presentation of five young adult IBD men and three women who failed to respond to standard medical therapy: this included prednisone, Disease Modifying Anti-Rheumatic Drugs (DMARDs) and Tumor Necrosis Factor inhibitors (TNF) termed biologics: predominantly prescribed as adalimumab and infliximab. The biologics are a class of drugs that inhibit inflammation at the cytokine, thymus, TNF factors, and interleukin levels. The author describes the *De novo* success both initially and in up to 5-years of long-term follow up by combining two or three FDA approved anabolic steroids. The long-term follow up was of two adults, cachexic men with multiple surgical resections and surgically induced Short Bowel Syndrome. It is the author's intent that reporting these initial and long-term successes with the Mixed Anabolic Treatment Program (MAT-P) will awaken interest in exploring a new directive in diagnosing, following, and treating IBD. There is evidence that IBD is a catabolic medical condition found to have lower levels of Total Testosterone (TT) and higher levels of Sex Hormone Binding Globulin (SHBG). The working hypothesis is that these hormonal changes are triggered by exposure to xenoestrogens that include man-made hormones in the environment: Bisphenol-A and-B, dioxin, DDT, glyphosates, and persistent organochloride pesticides (POP) ingested with food. Just as with *in vivo* natural estrogens, these man-made xenoestrogens have varying affinities to first attach to the Androgen-Receptor on the cell wall. They traverse the cytoplasm to attach to the Estrogen Receptor-alpha (ERα) and Estrogen Receptor-beta (ERβ) on the nuclear membrane. This proteins propagated by nuclear mRNA and DNA are abnormal, inflammatory and in time can produce an autoimmune reaction. This process can worsen over time and overwhelm the anabolic processes attempting to maintain homeostasis. There is evidence of a reproducible serum biomarker for IBD termed the Free Androgen Index (FAI). The host conversion from anabolic to catabolic is observed to parallel the FAI; the ratio of Total Testosterone divided by Sex Hormone Binding Globulin. The Total Testosterone (TT) represents the total anabolic potential of the host while the Sex Hormone Binding Globulin (SHBG) is seen to represent the influence of total *in vivo* estrogen and externally derived *in vitro* xenoestrogen activity. A low FAI is proposed to be a diagnostic biomarker of inflammatory disease in both sexes. Progressive increases in the FAI is a biomarker of observed improvement. There is evidence that medical treatment in the Mixed Anabolic Treatment Protocol (MAT-P) described herein successfully uses naturally occurring anabolic steroids of testosterone and nandrolone to raise the serum total testosterone to therapeutic ranges. Used concurrently with stanozolol, the first derivative of dihydrotestosterone, the Mixed Anabolic Treatment Protocol (MAT-P) blocks the host liver production of SHBG. Observations reported herein confirm that bringing the biomarker, FAI, into normal and supra-physiological range corresponds to recovery in cases of IBD where all medical and surgical standard treatments had been exhausted. This review encourages the gastroenterologist and the surgeon to see IBD as an environmental mediated hormonal catabolic process; that the directed addition of anabolic steroids resets the hormonal biomarker, corrects the host homeostasis hormonal milieu, and thereafter, reverses the inflammatory nature of the dominating estrogenic hormones. The paradigm shift utilizes hormonal anabolic medications to thwart the xenoestrogen burden on the host without which, the host can direct its energy to repair and 'finding the cure.'

Biography: Refer to Page No 36.

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Yoshiaki Omura

New York Medical College, USA

Non-invasive, early diagnostic methods of various cancers and their metastases. Role of coexisting contributing factors of Human papilloma virus-Type 16 and *Toxoplasma gondii* infection and strong, electromagnetic field, etc. for rapid cancer development (some cancers may be infectious): Safe, effective treatment of various cancers using non-invasive, safe, effective, simple, individualized treatment by combined use of optimal dose of vitamin D3 and thymus gland stimulation

As non-invasive, early diagnostic methods of various cancers, we developed following 5 new methods: 1) visible and invisible changes appearing at different parts of the face including eyebrow, alars of the nose, upper and lower lips, and thymus gland representation areas on the face & hands 2) One-Page Mouth, Hand, and Foot Writing Form, cancer can be detected without knowing anything about the patient, 3) detection of cancer from rapidly changing QRS Complex of ECGs, 4) abnormal thymus gland representation area, 5) bone marrow representation areas of each side of the face. Recently we found in the presence of multiple cancer-contributing factors, cancer can develop in less than few months. The important common factors we found is co-existence of Human Papilloma Virus-Type 16 (HPV-16) and single-cell *Toxoplasma gondii* as well as frequent strong electromagnetic field (EMF) exposure from cellular phone & strong negative underwear, metal products on the body surface, and toxic drinks & food. Therefore, some of rapidly developed cancer is infectious. Ideal treatment of cancer has to eliminate their infection in addition to inhibition or killing or elimination of cancer cells. We succeeded in developing ideal cancer treatment by combined use of 8 unique, beneficial effects of optimal dose of vitamin D3 and 50-time manual stimulation of thymus gland representation area on the back of L-hand was found to be most safe, effective treatment. Among these 8 unique, beneficial effects, most important ones are 1) very safe, strong anti-cancer effects including significant decrease in 8-OH-dG, which is proportional to DNA mutation and is required for cancer growth, 2) its improved circulation, 3) it has significant urinary excretion effects of Viruses, Bacteria, Fungi, single-cell parasites, and Toxic substances, including Asbestos & metals such as Hg, Pb, & Al, 4) about 15-time increase of Thymosin α1 which is increased in all the thymus gland representation areas on different parts of the body. As one of the most important & significant effects of 50-time manual stimulation of back of L-hand which increases Thymosin α1, 15 times of normal amount which is usually about 20ng. Even without stimulating any other thymus gland representation areas including main thymus gland at manubrium & rest of thymus gland representation areas at rest of the body are all activated and produced same changes as stimulated side of L-hand. Thymosin α1 is one of main hormones excreted from thymus gland and it is well-known that it has very significant anti-cancer effects. When we do this combined stimulation, total amount of increase in Thymosin α1 becomes total of about 30 times of non-stimulated condition. Since effect of optimal dose of vitamin D3 lasts about 6~8 hours, thymus gland stimulation effect also lasts close to similar time duration. We have been using every 6~8 hours, depending on the patient. So far, anti-cancer effect was very significant. We hope many advanced cancer patients can be saved by this method. So far, we are able to save number of terminal cancer patients which could not improved by standard cancer hospital treatment.

Biography

Yoshiaki Omura received Oncological Residency training at Cancer Institute of Columbia University & Doctor of Science Degree through research on Pharmaco-Electro-Physiology of Single Cardiac Cells *in-vivo* and *in-vitro* from Columbia University. He researched EMF Resonance phenomenon between 2 identical molecules for non-invasive detection of molecules, at Graduate Experimental Physics Department, Columbia University, for which he received US patent.

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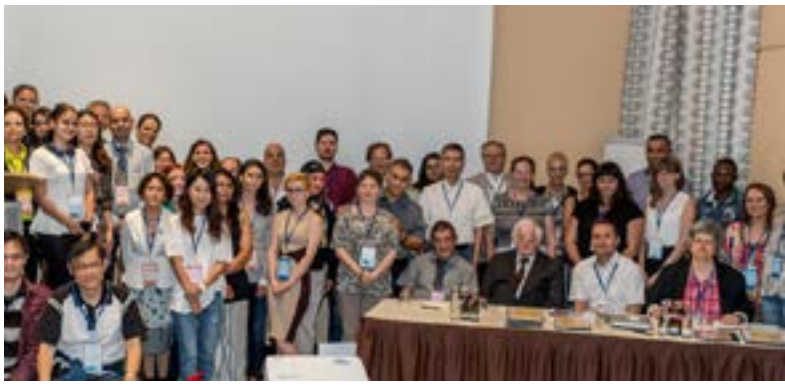

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G M Anantharamaiah

University of Alabama at Birmingham, USA

Apolipoprotein E mimetics dramatically reduce plasma cholesterol levels in several animal models

Analogous to apolipoprotein (apo) E, apoE mimetic peptide is a dual domain peptide, containing receptor binding domain from apoE (LRKLRKRLLR, [hE], residues 141-150), linked to 18A the lipid-associating peptide. The resulting peptide, Ac-hE18A-NH_2, reduces plasma cholesterol in several animal models and possess anti-inflammatory properties which are independent of the effect on plasma cholesterol. To enhance the cholesterol-reducing ability, we synthesized several analogs of this peptide with fatty acyl chains of different length to LRRLRRRLLR-18A-NH_2 ([R]hE18A-NH_2) to produce Ac-Aha-[R] hE18A-NH_2, Octanyl-, Oleyl-, Palmityl- and Myristyl-[R]hE18A-NH_2. The modified peptides were much more effective in reducing plasma cholesterol in apoE null mice. Myristyl-peptide analog was the most effective. This analog was also most effective in apoE null mice fed a Western diet, capable of reducing plasma cholesterol from 900mg/dL to almost undetectable amount of plasma cholesterol in 5h. Plasma cholesterol levels in cynomolgus monkeys fed a Western diet was reduced by the Myristyl-analog in a dose-dependent manner. A single dose maintained plasma cholesterol and low-density lipoprotein (LDL) levels below baseline even after one week. However, plasma HDL levels were increased compared to baseline levels. Considering the peptide Ac-hE18A-NH_2 has undergone Phase 1 clinical trials in humans, the new and highly active analogs are expected to exhibit enhanced potency with lower doses in humans.

Biography

GM Anantharamaiah is a Professor in the Department of Medicine. He received his BS degree in 1967 from Bangalore University, India and his MS degree in 1969 from Bangalore University, as well. He completed his PhD degree in 1978. He has published more than 190 research papers and has several patents to his credit.

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Vaidya Balendu Prakash

VCPC Research Foundation, India

Observational clinical study to note the impact of the ayurvedic mineral complex in pancreatitis patients

Pancreatitis, an inflammatory disorder of pancreas, is mainly categorized into acute and chronic form. While chronic pancreatitis is irreversible and progressive, even a single attack of acute pancreatitis can convert into chronic pancreatitis. The disease brings substantial physical, emotional, psychological and financial burden to patients and their families due to its unpredictable nature. The disease is also associated with early mortality. India reportedly has the highest incidences of pancreatitis. Owing to the limitations of modern medicine, many patients of pancreatitis turn to alternative and complementary medicine. In India, it is more so where many alternative systems of medicines are officially recognized as independent medical systems along with modern medicine. An Ayurvedic Mineral Complex (AMC) developed by an Ayurvedic physician brought miraculous recovery in a terminally ill patient of pancreatic cancer. The same formulation also showed repeated success in treating Pancreatitis patients. The practice was subjected to documentation following Good Clinical Practice guidelines since 1997. Till date, nearly 620 well-diagnosed cases of pancreatitis have opted for this treatment at their own expense. Results indicate that AMC brings significant reduction in weight loss, number of attacks and hospitalizations and improves weight. The patients were off all pancreatic enzymes after the start of Ayurvedic treatment. Safety studies have shown that AMC causes no Grade II toxicity. In an experimental study, AMC has shown a significant protective effect in animal models of pancreatitis. There is strong *Prima facie* that AMC brings sustainable complete relief to pancreatitis patients. However, further studies are required for understanding and development of the formulation.

Biography

Vaidya Balendu Prakash, born on March 14, 1959, at Meerut. He completed his university graduation in Science and Ayurveda medicine, learned applied aspects of Rasa Shastra from his father Vaidya Chandra Prakash, set up Vaidya Chandra Prakash Cancer Research Foundation (SIROs) to carry basic research in Ayurveda under the patronage of Late Dr KR Narayanan (Former President of India). He is the first and only Ayurvedic physician to get the Life Membership of Indian Co-operative Oncology Network (ICON) and is a member of the International Headache Society (IHS), UK.

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Hossein Akhondi

University of Nevada, USA

Diagnostic approaches and treatment of eosinophilic esophagitis: A review article

Eosinophilic Esophagitis (EoE): A condition that involves eosinophilic influx into the esophagus epithelium. It affects both children as well as adults. Adults present with dysphagia whereas children with vague abdominal complaints were being affected. The clinical symptoms, as well as pathological features of EoE and gastroesophageal reflux disease, are similar. Since eosinophilia in the esophagus is a non-specific finding, the clinical presentation in conjunction with endoscopic findings and pathology is crucial in determining a differential diagnosis. Because of the similarity between EoE and reflux, reflux should be excluded by using high proton pump inhibitors or through evidence of a normal pH by esophageal testing prior to treatment with an elimination diet or orally swallowed inhaled steroids.

Biography

Hossein Akhondi, MD is a practicing Internist in Las Vegas, NV. He is graduated from Iran University of Medical Sciences in 1995 and has been in practice for 22 years. He completed a residency at Mercer University School of Medicine. He currently practices at Mountain View Internal Medicine Associates and is affiliated with Mountain View Hospital. He accepts multiple insurance plans including Aetna, Medicare, and Humana. In addition to English, his practice supports these languages: Hindi, Urdu, Farsi/Persian, Punjabi, and Spanish.

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Notes:

International Conference on
GASTROINTESTINAL CANCER AND THERAPEUTICS
4th World Congress on &
DIGESTIVE & METABOLIC DISEASES
26th Annual Congress on &
CANCER SCIENCE AND TARGETED THERAPIES
October 29-30, 2018 | San Francisco, USA

A Craig Lockhart

University of Miami, USA

Front-line chemotherapy for metastatic gastroesophageal cancers: A first-line study of FOLFIRINOX for patients with advanced gastroesophageal adenocarcinoma

Statement of the Problem: Esophageal and gastric cancers continue to pose a significant burden of morbidity and mortality globally. Presently, gastric cancer is the fifth most common cancer worldwide, and the third leading cause of cancer death. Esophageal cancer, though less common, has a striking mortality rate, placing it as the sixth leading cause of cancer-related deaths worldwide. Unfortunately, many patients with these cancers present at an advanced, incurable stage. Standard first-line regimens for patients with metastatic gastroesophageal adenocarcinomas have an approximate 40% objective response rate and only provide patients with a survival of less than 1 year.

Methodology and Theoretical Orientation: FOLFIRINOX chemotherapy has been used in first-line therapy in other GI cancers (i.e pancreatic and CRC) with impressive efficacy signals. This is a Phase II study of first-line combination chemotherapy with FOLFIRINOX (5-FU, irinotecan, and oxaliplatin) in patients with advanced gastroesophageal adenocarcinomas (NCT01928290).

Findings: This study enrolled 58 patients. The response rate with FOLFIRINOX was 78% in all patients. Median progression-free survival was 11.9 months, and median overall survival was 17.4 months.

Conclusion and Significance: Metastatic gastroesophageal cancers are increasing in incidence and are incurable. The FOLFIRINOX chemotherapy regimen may provide patients benefits over standard approaches. The FOLFIRINOX study results, as well as the current state-of-the-art treatment for gastroesophageal cancers, will be discussed in detail.

Biography

A Craig Lockhart is the Division Chief for Medical Oncology at the University of Miami School of Medicine, Sylvester Comprehensive Cancer Center (SCCC). He has over 15 years of early phase clinical trial experience where he has been the PI of over 100 Phase I, II and III trials. His own research focuses on Phase I/II clinical trials of novel therapeutics applied to gastrointestinal cancers.

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Nilay Sethi

Dana Farber Cancer Institute, USA

Comparative molecular analysis of gastrointestinal adenocarcinomas yields novel therapeutic strategies

Combined, adenocarcinomas of the esophagus, stomach, colon, and rectum account for a devastating 1.4 million deaths each year worldwide. Conventional treatment approaches currently offer modest benefit, inspiring our effort to identify previously unrecognized biological mechanisms underlying the pathogenesis of gastroesophageal (GE) and colorectal (CR) cancers. To better understand these cancers, we analyzed 921 adenocarcinomas of the esophagus (n=79), stomach (n=383), colon (n=341) and rectum (n= 118) obtained from the fresh frozen tissue by The Cancer Genome Atlas Network using six molecular platforms. We uncovered five molecular subtypes that largely transcended anatomic boundaries including Epstein-Barr Virus (EBV) positive and Hypermutated tumors (HM), which further substratified into MSI and Hypermutated-SNV (HM-SNV). The remaining two groups were distinguished by presence or absence of extensive SCNAs. Chromosomal instability (CIN) tumors exhibited marked aneuploidy, a feature that was essentially absent in the Genome stable (GS) subtype. Evaluating the anatomic distribution revealed that HM tumors primarily occupied the central part of the GI tract in the distal stomach and proximal colon, whereas CIN tumors were more prevalent in the anatomic extremes, namely, the esophagus and distal colon/rectum. Inspired by recent clinical advances in immunotherapy, we studied associations between our molecular groupings and key immune features. EBV+ were enriched for gene expression scores associated with CD8+ T-cells, M1-macrophages, and IFN-γ signatures. MSI tumors showed the next greatest IFN-γ signature and displayed diverse immune signatures depending on the tissue of origin of translational importance, an attenuation in HLA/antigen presentation and significant elevation in NK-cell gene expression was found in CR HM-SNV. Blockade of inhibitory signals or stimulation of activating cues can tip the balance in favor of cytotoxic endogenous NK activity, which may be a therapeutic option for patients with HM-SNV tumors. Overall, these findings further the rationale to develop innovative therapeutic strategies that recruit and reinvigorate the host immune system to battle cancer.

Biography

Nilay Sethi earned his PhD from the Molecular Biology Department at Princeton University in 2010 and his MD at Rutgers Robert Wood Johnson Medical School in 2012. He completed his internal medicine training at the University of California San Francisco (UCSF) in 2014 and medical oncology fellowship at the Dana-Farber Cancer Institute in 2017. His work in cancer metastasis has led to publications, including a landmark study on Jagged1 in bone metastasis, and yielded numerous awards. He has now focused on better understanding the molecular mechanisms underlying gastrointestinal malignancies with the hope of translational advances that will improve outcomes in patients suffering from these devastating diseases.

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Vinod Nikhra

NDMC Medical College and Hindu Rao Hospital, India

ACS-HOMS: The aberrancy in CNS signals and other factors related to altered homeostasis, obesity and metabolic syndrome

Introduction: 'The obese-obese world': The obesity and metabolic syndrome (MetS) are a global epidemic of such magnitude that the today's health scenario can be summed up as the 'Obese-obese World'. Obesity and MetS deteriorate the quality of life and alter course of various chronic diseases, and on their own, are risk factors for diabetes, hypertension, cardiovascular disease and stroke, neurological degenerative diseases and cancers. Modern day lifestyle drives for excess calorie intake, comparatively reduced energy expenditure and storage of surplus energy in adipose tissue, an accentuated evolutionary need to fill body nutrients stores, leading to obesity, appended by pathophysiological alterations termed MetS.

The regulation of energy intake: Specialised neurons in hypothalamus and brainstem primarily regulate energy homeostasis, food intake, and body weight, and integrate multiple peripheral metabolic inputs, such as nutrients, gut-derived hormones, and adiposity-related signals. There are several neuropeptides involved, including melanin-concentrating hormone (MCH) and the orexins. An abnormal alteration in ghrelin and leptin levels can lead to weight gain and obesity. Increase in adipose tissue leads to overproduction of leptin and hypothalamus resistant to leptin action. The reward circuitry involves interactions between several systems including opioids, endocannabinoids, serotonin, and dopamine. The obese individuals appear to have abnormalities in dopaminergic activity, and an imbalance in the brain circuits promoting reward seeking and those governing cognitive control leads to an overriding stimulus to feeding, even in the absence of an energy deficit. Dorsal striatum is hyperactive in obese and may contribute aberrancy of satiety signals. The genetics involving various mutations contribute up to 70% towards a person's vulnerability to obesity.

The regulation of energy expenditure: Energy is consumed in processes of physical activity, basal metabolism, and adaptive thermogenesis, which are modulated by the brain, especially the hypothalamic melanocortin system. Brown adipose tissue (BAT) plays a major role in thermogenesis. Central regulation of BAT thermogenesis is dependent on sympathetic outflow to BAT. Norepinephrine released from sympathetic nerve terminals binds to β3-adrenergic receptors on adipocytes in BAT to promote enhanced thermogenesis. In addition, many hormonal and nutrient signals, such as glucose, insulin, leptin, and GLP-1, also influence sympathetic outflow to BAT.

Conclusion: Fallouts of surplus energy storage: The obese subjects with BMI >30 show atrophy in the frontal lobes, anterior cingulate gyrus, hippocampus, and thalamus. MetS affects various cognitive domains including executive functioning, processing speed, and overall intellectual functioning. There is impaired vascular reactivity, endothelial dysfunction, neuroinflammation, oxidative stress and altered brain metabolism.

Biography

Vinod Nikhra is a consultant and faculty at NDMC Medical College and Hindu Rao Hospital, a multispecialty, thousand bedded, public hospital, New Delhi. He is an MD in Internal Medicine and qualified and trained in nephrology, endocrinology, and cardiology, and hospital management, and Fellow of International Medical Sciences Academy and Royal Society of Medicine, London. He has been Editor of "Madhya: the midage", the official Journal of Association for Health in Middle-Aged, on Editorial Board of Open Access Journal of Gerontology and Geriatric Medicine, on Reviewer panel of, among others, the Family Practice, an Oxford University medical journal and International Journal of Obesity. He is Author of four books, which include his widely acclaimed books, 'Aging slowly, Living longer' and 'The Anti-obesity Guide', and over 60 papers in international, national and other journals, some of which are available on www.researchgate.net. He has travelled widely and participated and spoken over in various international Conferences. Some of his talks are available on YouTube.

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Michael W Retsky

Harvard TH Chan School of Public Health, USA

Perioperative use of NSAID ketorolac might prevent early relapses in breast and other cancers including colon

A bimodal pattern of hazard of relapse among early stage breast cancer patients has been identified in multiple databases from US, Europe and Asia. We are studying these data to determine if this can lead to new ideas on how to prevent relapse in breast cancer. Using computer simulation and access to a very high quality database from Milan for patients treated with mastectomy only, we proposed that relapses within 3 years of surgery are stimulated somehow by the surgical procedure. Most relapses in breast cancer are in this early category. Retrospective data from a Brussels anesthesiology group suggests a plausible mechanism. Use of ketorolac, a common NSAID analgesic used in surgery was associated with far superior disease-free survival in the first 5 years after surgery. The expected prominent early relapse events in months 9-18 are reduced 5-fold. Transient systemic inflammation accompanying surgery (identified by IL-6 in serum) could facilitate angiogenesis of dormant micrometastases, proliferation of dormant single cells, and seeding of circulating cancer stem cells (perhaps in part released from bone marrow) resulting in early relapse and could have been effectively blocked by the perioperative anti-inflammatory agent. If this observation holds up to further scrutiny, it could mean that the simple use of this safe, inexpensive and effective anti-inflammatory agent at surgery might eliminate early relapses. We suggest this would be most effective for triple negative breast cancer and be especially valuable in low and middle income countries. Similar bimodal patterns have been identified in other cancers such as lung, prostate, osteosarcoma, head and neck, nasopharyngeal, esophageal, and pancreatic suggesting a somewhat general effect. Regarding colon cancer, it seems that this effect is active after partial hepatectomy for treatment of hepatic malignancies but less clear what is happening after primary colon cancer resection.

Biography

Michael Retsky (PhD in Physics from University of Chicago) made a career change to cancer research thirty years ago. He is Research Associate at Harvard TH Chan School of Public Health and Honorary Reader at University College London. He was on Judah Folkman's staff at Harvard Medical School for 12 years. He is the Editor of a Springer-Nature book on the breast cancer project published in July 2017. After the diagnosis of stage IIIc colon cancer in 1994, he was the first person to use what is now called metronomic adjuvant chemotherapy. He is a founder and for 10 years was on the Board of Directors of the Colon Cancer Alliance. He has published more than 60 papers in physics and cancer.

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George Vasmatzis

Mayo Clinic, USA

Testing HER2-related therapeutic strategies in GI cancers guided by integrated genomics

Many somatic mutations have been detected in GI cancers, leading to the identification of some key drivers of disease progression, but the involvement of large genomic rearrangements has often been overlooked. Chromosomal rearrangement detection allows coverage of the entire genome as opposed to narrowly focusing on selected genes. It is far less prone to false positives and can potentially identify driving rearrangements/fusions within pathways that may be therapeutically targetable. Due to tumor-specificity of the breakpoint-junctions, it can be used for defining clonal relationships and allows for designing individualized diagnostic tests for monitoring a patient's disease progression. We performed mate-pair sequencing (MPseq) on genomic DNA and RNAseq on mRNA from several patients with pancreatic adenocarcinoma, cholangiocarcinoma and colorectal cancer to identify genome-wide rearrangements. We found a small number of potentially targetable amplifications and fusions including four cases with ERBB2 amplification. 3D micro cancer modeling was performed followed by a drug screen informed by the genomic analysis. Finally, dose-response curves (including IC_{50} values), were generated after measuring cellular ATP. Significant responses were noted for all Her2 targeted therapies. The combined genomic/micro cancer analysis pointed towards the possibility that these patients have a Her2 activated pathway and could benefit from Her2 targeted therapies.

Biography

George Vasmatzis is a Consultant in the Department of Molecular Medicine and a member of the Mayo Clinic Cancer Center, as well as the co-director of the Biomarker Discovery Program, within the Center for Individualized Medicine. His research program consists of bioinformatics specialists, molecular biologists, epidemiologists, and pathologists. His team has demonstrated success in discovery and translation of several biomarkers as well as developing evidence-based models that should help clinicians stratify (cancer) patients in order to provide each individual with the appropriate care. With the recent advances in Next Generation Sequencing (NGS) technologies his laboratory has been engaging in massive sequencing to scan the genome of cancer cells for abnormalities that can be used for clinical purposes such as diagnosis and stratification of patients for optimal treatment. Published papers in the journal of Clinical Oncology, Cancer Research, and BLOOD further demonstrate their discovery, validation, and translation capabilities.

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Andrei L Gartel

University of Illinois, USA

Targeting FOXM1 in colon and liver cancer

Colorectal cancer (CRC) is the third most commonly diagnosed cancer and the second leading cause of cancer-related death in the United States. Therefore, development of novel therapeutic strategies for the treatment of CRC is extremely important. FOXM1 overexpressed in the majority of CRC and overexpression of FOXM1 plays a critical role in colorectal cancer. Since the FOXM1 regulatory network is a major predictor of adverse outcomes in human cancers, inactivation of FOXM1 by the FOXM1 inhibitors an attractive treatment strategy. Nucleophosmin (NPM) belongs to the nucleophosmin/nucleoplasmin family of chaperones, which are ubiquitously expressed in mammalian cells. FOXM1 interacts with NPM in human cancer cells including CRC cells and NPM knockdown in human cancer cells led to significant down-regulation of FOXM1. Our data suggest that in human cancer cells NPM interacts with FOXM1 and their interaction is required for sustaining the level and localization of FOXM1. We identified two compounds that inhibit NPM/FOXM1 interaction and suppress FOXM1 expression in CRC cell lines. In addition, these compounds synergize with 5-FU in HCT116 CRC cells. NPM consists of pentamers that dimerize into a decamer. The compounds are predicted to bind at two sites on NPM homo-oligomerization domain and they would likely block NPM oligomerization. Therefore, by disrupting monomer-monomer interactions, they are also precluding binding of NPM and FOXM1. We hypothesize that since FOXM1 contributes to the progression and metastasis of CRC, targeting FOXM1 with small molecules will improve treatment outcomes for CRC patients.

Biography

Andrei L Gartel, PhD, is an Associate Professor in the Department of Medicine at the University of Illinois at Chicago and is the academic editor of PLOS ONE. He is the author of 89 peer-review publications that include more than 20 reviews. He has more than 10,000 citations and his h-index is 40. His scientific interests include cancer, cell cycle, protein-protein interactions, regulation of CDK inhibitor p21 and regulation of oncogenic transcription factors FOXM1, and c-Myc. Specifically, his lab is interested in the identification of new FOXM1 inhibitors. He received his funding from NIH, DOD and private companies/foundations.

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