6(i): Clinical Medicine

Session Chair
Dr. Eugene P. Goldberg
University of Florida, USA

Session Co-Chair
Dr. Sudhakar Akul Yakkanti
Boys Town National Research Hospital, USA

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Title: Sam-Pointed Domain Ets Transcription Factor-1 (SPDEF-1, a.k.a. PDEF-1) is a Tumor Metastasis Suppressor and its Mechanism(s) of Action

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doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Intratumoral chemotherapy (ITC) as adjunct to standard therapy in NSCLC iiia-iv prolongs life

Wolfgang Hohenforst-Schmidt
Coburg Klinic, Germany

The efficacy of conventional intravenous cancer chemotherapy is severely limited by systemic drug toxicity. Statistics for the past 20 years indicate little progress in reducing cancer mortality except for distinctive genetically defined subtypes. Reported here are studies showing the efficacy of *intratumoral chemotherapy* (ITC) as a debulking tool in central tumors applied worldwide in more than 370 published patients over the last decade. *ITC* has already shown in few observational studies that a surplus of median survival can be achieved if this method is as an adjunct to standard options like reduced intravenous chemotherapy, external or internal radiotherapy even in patients with poor performance status: Cancer drugs are injected directly into the tumor for central tumors or transthoracically for peripheral cancer site and by an EBUS-system for involved mediastinal lymph nodes. In more than 60 published patients (inoperable IIIa – IV) a surplus of up to 77% in median survival (MS) in comparison to expected MS according to UICC 7 data was achieved when ITC was used as an adjunct to standard therapy. Superdoses of cytotoxic drugs may thereby accomplish rapid tumor cell killing without systemic toxic complications but also involved lymph nodes - in many studies the hallmark of local recurrence – could be treated directly and specifically. This new paradigm promises to significantly reduce lung cancer morbidity and mortality without the toxic complications associated with conventional systemic chemotherapy. It maybe not only considered in palliative situations but also as preoperative therapy according to the results in animal studies.

Biography

Dr. Wolfgang Hohenforst-Schmidt works as a senior physician executive in the field of interventional pulmology including chest oncology, interventional cardiology and intensive care medicine since more than one decade. He is author of the national guideline commitee on Pulmonary Hypertension (Dtsch Med Wochenschr 2010; 135: S102-115). In interventional pulmology he published new methods like perthoracical endopulmonary ultrasound to guide peripheral cancer biopsies (49th Congress of the German Society of Pulmology (DGP) 2008, Lübeck, P79) and reported for the first time surprising survival rates in NSCLC-patients following an interventional program that used controlled submaximal physical exercise as adjunct treatment to standard therapy (Medical Tribune 2010; 31/32: S16). On the 16th World Congress of Bronchology in Budapest he presented surprising preliminary data on survival of patients treated with ITC in combination with intravenous chemotherapy (16th WCB 2010, Budapest, A-0190).

Eugene P. Goldberg, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Endobronchial Intratumoral Chemotherapy (EITC): A New Modality for Palliation and Potential Curative Therapy of NSC Lung Cancer

Eugene P. Goldberg[1], Wolfgang Hohenforst-Schmidt[2] and Seyhan Celikoglu[3]

[1]Colleges of Engineering & Medicine, University of Florida, USA
[2]Klinikum Coburg, Germany,
[3]University of Istanbul Medical Center, Turkey

Lung cancer remains the most deadly and most difficult cancer to treat effectively. The standard of care for conventional treatment; radiation, systemic chemotherapy and surgery is relatively ineffective for long term survival. CDC statistics indicate a 75% increase in lung cancer mortality during the past 20 years. New concepts for enhancing quality of life and for prolonged survival are needed. Reported here is progress for a new therapeutic paradigm, intratumoral (IT) chemotherapy, a novel localized treatment modality. The procedure, endobronchial intratumoral chemotherapy (EITC) involves direct intratumoral drug injection via a needle bronchoscope. A superdose of drug is thereby made to perfuse the tumor mass and achieve rapid tumor necrosis and massive tumor cell killing. Because of the localized drug delivery, there are no systemic toxic complications with cisplatin or mitoxantrone that are normally associated with conventional IV chemotherapy. Palliation and prolonged survival have been observed clinically for EITC, especially for patients presenting with significant airways obstruction. Collaborative clinical studies has been conducted with Dr. Celikuglu, who has pioneered EITC in Istanbul, and with Dr. Hohenforst-Schmidt in Germany. Favorable clinical outcomes for EITC have now been observed for hundreds of NSCLC patients. In parallel preclinical IT chemotherapy research in Florida, using a murine Lewis lung carcinoma, mitoxantrone-loaded albumin nanomesospheres afforded prolonged IT tumoricidal activity and evidence for systemic tumor-specific immune response. Additional research indicates that IT neoadjuvant chemotherapy followed by resection of the necrotic tumor mass may afford a curative response. It is reasonable to conclude that IT chemotherapy represents an important new approach to improved lung cancer treatment.

Biography

Dr. Goldberg FAIMBE, FBSE, joined the faculty of the University of Florida as the Biomedical Program of Excellence Professor in 1975. At Florida, as part in the Departments of Chemistry and Materials Science & Engineering, he helped establish intramural graduate programs in Polymer and Biomedical Sciences. He is now also affiliated with the University's Cancer Center and the Departments of Biomedical Engineering, Pulmonology, and Pharmacology & Therapeutics. His biomedical research interests and activities for the past 35 years have been diverse with strong focus on localized chemotherapy by direct intratumoral drug injection. Pioneering cancer therapy studies were initiated in 1976 as a Visiting NIH Scientist and marked by a seminal 1978;38:1311 Lung Cancer paper on IT Chemoimmunotherapy. Subsequent research was devoted to enhancement of intratumoral chemotherapy using drug-loaded albumin and DNA nanomesospheres as reviewed in JPP 2002;54;159-180. Recent clinical research has been focused primarily on bronchoscopic intratumoral injection of chemotherapy with Drs. Seyhan and Firuz Celikoglu and Dr. Wolfang Hohenforst-Schmidt as reported in Cancer Therapy 2008;6:545-552 and JPP 2010;62:287-295. Dr. Goldberg is the senior author of more than 425 published and presented papers and is on the editorial boards of numerous journals.

Sudhakar Akul, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Extra cellular matrix derived endogenous angioinhibitor tumstatin and its mechanism(s) of action

Sudhakar Akul

Boys Town National Research Hospital, USA

Cancer is currently one of the most prevalent causes of human deaths in the world. Current therapeutic options aim only to slow the progression of cancer disease. Therefore, a renewed effort must be made to identify relevant endogenous cancer inhibitors that could be exploited as therapeutic drugs. We identified several endogenous anti-cancer molecules, which are released from extracellular matrix (ECM) into the blood circulation of cancer patients. Several of these endogenous circulating molecules were cloned and identified as angioinhibitors of tumor growth. These endogenous angioinhibitory proteins bind to the cell surface integrins and transduce the signalling mechanisms & regulate angiogenesis. Thus, integrins serve as transmembrane linkers between the ECM and cytoskeleton for outside-in signalling. One such endogenous circulating molecule, tumstatin, a 28-kDa protein from the C-terminal non-collagenous (NC1) domain of alpha3 type IV collagen was identified by us as an inhibitor of angiogenesis (Science 2002; PNAS 2003). Tumstatin interacting with alphaVbeta3 integrin and inhibits activation of focal adhesion kinase (FAK), phosphatidylinositol 3-kinase (PI-3K), serine/threonine kinase (Akt/protein kinase B), mammalian target of rapamycin (mTOR) and prevents dissociation of eukaryotic translation initiation factor 4E (eIF4E) from 4E binding protein (4E-BP1) leading to the inhibition of Cap-dependent translation in proliferating endothelial cells. Recently, we also demonstrated that tumstatin inhibits hypoxia induced pro-inflammatory cyclo-oxygenase-2 (COX-2) expression via FAK/Akt/NFkB pathway, leading to decreased tumor angiogenesis and tumor growth in an alpha3beta1 integrin dependent manner (Blood 2007; J Canc Sci Ther 2009). At present my laboratory is studying to understand four such endogenous angioinhibitor molecules derived from type IV collagen that include tumstatin, arresten, combostatin and hexastatin which are involved in cell signalling and the way these proteins control adhesion and migration of endothelial cells in pathological processes including tumor angiogenesis.

Biography

Sudhakar Akulapalli (Akul) is the founder Director of Cell Signaling, Retinal and Tumor Angiogenesis Laboratory at Boys Town National Research Hospital, Associate Professor at Creighton University School of Medicine and University of Nebraska Medical Center, Omaha, NE, USA. He did his postdoctoral training at Harvard Medical School, Boston, MA, USA (2003). He has received Ph.D (2001), M.Phill (1997) and M.Sc (1995) degrees in life sciences from University of Hyderabad; and B.Sc in Biology from Silver Jubilee College (APRDC) Kurnool, SK University (1993) from India. He received President's fellowship (1992), GATE (1996) and CSIR (2007-2000) fellowships from Government of India. He received Mahindra & Mahindra Educational Award (2000) and Young Clinical Scientist Awards from Flight Attendant Medical Research Institute (FAMRI) in 2007 and 2010. He also received Bio-Bio Young Scientist Award from OMICS publishing group; Michael A. O'Connor Young Investigator Award; RO1 grant Award from NIH/NCI and Research Scholar Grant Award from ACS (2010). He is serving as AIBS/NIH-RO1 Grant reviewer for DT study section. He has published more than 35 research articles in several top journals including Science, Cancer Cell, JCI, Blood, PNAS, Gastroenterology, Cancer Research, JBC, IOVS, JCST etc. He is serving as an Executive Editor, Editor and Editorial board member of reputed journals and is serving as a reviewer for 21 scientific journals including JCI, Blood, Circulation, Circulation Research, Cancer research, Clinical Cancer research etc. He was honored by giving a position as Keynote Speaker, Chairman, Co-chairman and organizing committee member for several international conferences including Bio-Bio-2009; Bio-Bio-2010; Anal-Bio2010; Biomarkers & Clinical Research 2010; Diabetes & metabolism 2010 etc.

Eun-Jung Kim, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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The association between Charlson Comorbidity Index(CCI) and the burden of cancer

Eun-Jung Kim

Cheju Halla University Department of Nursing, Republic of Korea

This study compared the association of comorbidity related with health outcomes by considering the fatality of cancer. Lastly, it will examine how the association on health outcomes differs according to patterns of associated diseases by types of cancer and will analyze the correlation with health outcomes. This retrospective, non-controlled and non-randomized study was conducted with 287 breast cancer patients, 273 colon cancer patients, 614 stomach cancer patients and 391 lung cancer patients. Using claim data, I measured comorbidity index. I used EDI claim data for calculating medical cost. Multiple regression and logistic regression model were utilized to investigate the effect of comorbidity on health outcomes as a total medical cost. This study controlled demographic characteristics and stage of cancer to estimate the influence of CCI on health outcomes. All statistical analysis was performed with sas 9.1. The effect of CCI measured with the medical records on the medical costs is higher CCI increased the medical cost of stomach cancer patients 1.05 and the cost of colon cancer patients 1.01. The breast cancer patients with COPD paid more medical cost than those without it and the increasing rate got lesser when CCI increased. And the colon cancer patients with DM paid more medical cost when the CCI got 1 point. But the lung cancer patients with COPD and CCI 2 point paid less than other patients. There are some differences according to comorbid diseases due to the characteristics of each cancers. The chronic comorbid were major factor to increasing medical cost and using medical resources. To prevent above mentioned disease, we must focused to check metabolic syndromes then preserve insurance financial health.

Biography

Eun-Jung Kim has completed her Ph.D at the age of 30 years from Korea University. She is the assist professor of Cheju Halla University. She has published more than 5 papers in reputed journals and serving as a member of repute.

Alberto A. Gabizon, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Therapeutics and Toxicology of Liposome-Based Anticancer Drugs

Alberto A. Gabizon

Shaare Zedek Medical Center and Hebrew University-School of Medicine, ISRAEL

Most of the currently used anti-tumor agents have problematic toxicities compromising efficacy, and often resulting in life-threatening events. Liposomes can provide effective control of the release rate and of the tissue distribution of many of these agents. These pharmacokinetic changes often have a major pharmacodynamic impact with attenuation of toxic effects and protection of sensitive tissues from dangerous and unwanted drug exposure. Polyethylene-glycol (PEG) coating of liposomes results in inhibition of liposome uptake by the reticulo-endothelial system and significant prolongation of liposome residence time in the blood stream. A hallmark of these long-circulating liposomal drug carriers is their enhanced accumulation in tumors. The mechanism underlying this passive targeting effect is the phenomenon known as enhanced permeability and retention (EPR) which has been described in a broad variety of experimental tumor types, and appears also to be a relevant phenomenon in human cancer. Developments in drug loading technology have improved the efficiency and stability of drug entrapment in liposomes, particularly with regard to anthracyclines, vinca alkaloids, and camptothecin analogs. Coupling the advances in liposome engineering such as pegylation with highly efficient drug remote loading techniques has resulted in robust formulations with great improvements in pharmacokinetics and pharmacodynamics over the conventional administration of cytotoxic drugs.

An example of liposome formulation with demonstrated clinical added value is pegylated liposomal doxorubicin (PLD), which has demonstrated clinically a favorable safety profile and proven efficacy against various malignancies and can be considered as the first anti-cancer nanomedicine approved for clinical use. The clinical pharmacokinetic profile of PLD is characterized by slow plasma clearance and small volume of distribution with drastic shifts (~1000-fold) from free doxorubicin. Based on preclinical studies, other formulations such as pegylated liposomal irinotecan hold promise to offer an important clinical edge in cancer chemotherapy. Another type of approach applicable to liposomal drug delivery combines the concept design of a stable and long-circulating liposome with chemical modification of a drug to provide a lipophilic prodrug with strong association to the liposomal bilayer. This is the case of a prodrug of mitomycin-C activated by thiolytic cleavage. Thiolytic cleavage takes place in the tissue micro-environment with negligible activation in plasma thus preventing drug activation and drug leakage in the blood stream and resulting in 3-fold decrease in toxicity when compared to treatment with free mitomycin-C.

Further to the passive targeting effect, the liposome drug delivery platform offers the possibility of grafting tumor-specific ligands on the liposome membrane for active targeting to tumor cells, and potentially intracellular drug delivery. Ligand-specific targeting may enhance tumor drug accumulation and reduce further the toxicity of liposome-delivered drugs in comparison to passively targeted systems.

Liposome-based systems offer a vast array of potential applications in the delivery of cancer chemotherapeutic agents. Provided liposome composition and drug entrapment are properly engineered, major changes in the pharmacokinetics and biodistribution can be obtained. Pharmacodynamic changes may result in a substantial improvement of the toxicity profile and in a significant enhancement of the therapeutic index of the entrapped drug. Although liposomal doxorubicin has already found a place in routine clinical use, the potential of liposomal drug delivery remains so far under-exploited.

Karl Kingsley et al., J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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High-risk human papillomavirus (HPV) screening and detection in normal, healthy patient saliva samples: a pilot cluster randomized study

Karl Kingsley and Deidre Turner

University of Nevada, School of Dental Medicine, USA

Human papillomavirus (HPV) is the primary etiological factor that transforms cervical epithelia into cancer. The presence of HPV in oral cancers suggests that HPV may play a similar role in transforming the oral epithelia. In addition to Epstein-Barr and Cytomegalovirus, new evidence has also revealed the frequent presence of high-risk HPV strains in breast carcinoma biopsies. Although epidemiologic studies suggest tobacco and alcohol use, and genetic predisposition are likely responsible for oral and breast carcinogenesis, concomitant HPV infection may be a significant factor that mediates growth and development. Although HPV may be transmitted from the oral cavity to the breast through direct contact, little evidence to date regarding oral HPV prevalence among health adults in the United States is available. The current study involved a non-invasive HPV screening of normal healthy adults at a US dental school, randomly selected to participate in a clustered pilot study. DNA was isolated from saliva samples and screened for HPV16 using qPCR. Chi-square analysis revealed the random patient sample was representative of the general clinic population with respect to gender, race and age ($p < 0.05$). Four patient samples were found to harbor HPV16 DNA, representing 3.9% of the total ($n = 102$); all four were female and Hispanic. This provides new information about oral HPV status, which may help to contextualize results from other studies demonstrating increasing oral cancer rates among females and minorities and in some geographic areas that may be associated with risk factors in addition to tobacco and alcohol use.

Biography

Karl Kingsley completed his PhD in 2001 and subsequently pursued postdoctoral studies at Stanford University in the School of Medicine, Division of Hematology. Dr. Kingsley is currently an Associate Professor of Biomedical Sciences at the UNLV School of Dental Medicine, where he teaches and directs an oral cancer research laboratory, specifically investigating high-risk HPV infection. He recently earned a Master of Public Health (MPH) in Occupational and Environmental Health and has published more than 25 papers in peer-reviewed journals. In addition, Dr. Kingsley is an avid supporter of the American Cancer Society in Las Vegas, Nevada.

Martins Thomas, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Molecular basis of anti-inflamatory strategies in cancer cachexia

Martins Thomas

Cardiothoracic Surgery Unit, College of Medicine of University of Lagos, Nigeria

Background: There are newer diagnostic and therapeutic armamentaria for pri mary lung cancer. Application of molecular genetics in lung cancer management is evolving rapidly. However, the traditional knowledge and practices that were applicable before the 1980s still hold sway in most developing countries.

Aims and Objectives: This research was conducted to highlight the staggeri ng gap in the current aetiopathology and management profile of primary lung cancers and to assess the readiness of developing world for the chal lenges of lung cancer management in the new decade.

Methods: We studied the patients referred to Lagos University Teaching Hospital with suspicion of primary lung cancer. We noted their bio-dat a, predisposing factors and final diagnosis on completion of investigations. We also noted the therapeutic modalities that were applied - especially the type of operation that was done for each of the patients.

Results: The research lasted 99 months beginni ng in October 1999 and 267 patients were enlisted. There were 148 males (55.4%) and 119 females (44.5%). Stage IV patients were 183 (68.5%) while only 3 patients were found at stage I. Histology showed squamous cell carcinoma in 27.7% of cases while adenocarcinoma constituted 64.0%. Curative surgery was performed for 13.1% while non curative surgery was performed for 16.5%. Correlation between smoking and malignancy was stronger among the males than the female patients.

Discusion and Conclusion: There is increasing incidence of primary lung cancers among non-smoking females.

Erwin Jannino O. Ybanez, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Nose bleed gone wild: Extramedullary plasmacytoma of the right nasal septum

Erwin Jannino O. Ybanez
Davao Doctors Hospital, Philippines

This is a rare case of extramedullary plasmacytoma (EMP) of the right nasal septum in a 25-year-old, Filipino, woman. She presented with recurrent episode of epistaxis and a mass in the right nasal cavity. Nasal endoscopy revealed a friable mass occupying the right anterior nasal cavity originating from the right lateral nasal wall superior and anterior to the inferior turbinate. Computed tomography of the paranasal sinuses showed a nipple-like structure projecting to the side of the nasal septum compatible with a vascularized polyp. The mass was completely removed endoscopically and histopathologic examination showed a densely packed tumor cells showing ovoid polygonal polychromatic and vesicular nuclei with moderate eosinophilic cytoplasm. Immunohistochemical staining showed positive for kappa and lambda light chains and negative for cytokeratin (CK) and leukocyte common antigen (LCA). Biopsy specimen was strongly immunoreactive to CD79a, MUM-1 and Ki67, consistent with EMP. Three months after initial polypectomy, the patient noticed recurrence of right nasal obstruction. A repeat CT scan of paranasal sinuses revealed right nasal mass almost entirely occupying the nasal cavity. Polypectomy and histopathologic examination of the specimen was still consistent with plasmacytoma. All diagnostic evaluation in this patient didn't show evidence of multiple myeloma. After removal of nasal mass, she received postoperative radiotherapy with total dose of 4500cGY to tumor bed. She remains disease free after six months. EMP of the nasal septum should be one of the differential diagnoses for nasal mass with history of recurrent epistaxis and nasal obstruction.

Jan Kotarski, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Evaluation of dendritic cells and RANTES in patients suffering from ovarian cancer

Jan Kotarski, Iwona Wertel and Wanda Rogowska
Medical University, Department of Oncological Gynaecology and Gynaecology, Lublin

The study was undertaken to evaluate Regulated on Activation, Normal T-cell Expressed and Secreted (RANTES) levels in the peritoneal fluid (PF) and plasma of patients with different stage, grade and histological type of ovarian cancer (n=73) or serous cystadenoma (n=32) in relation to PF and peripheral blood (PB) myeloid and lymphoid dendritic cells (DCs). The PF and plasma level of RANTES was detected using ELISA assay. DCs were estimated using flow cytometry. The following directly conjugated mAbs were used: anti-BDCA-1 (CD1c) FITC, anti-BDCA-2 (CD303) FITC and anti-CD19 CyChrome, anti-CD123 PE.

The percentage of myeloid DCs was significantly lower in the PF of patients with ovarian cancer (0.64%) than in women with benign tumors (7.76%). In contrary, the percentage of lymphoid DCs was higher in the PF of patients with malignant disease (0.66%) than in the reference group (0.20%).

The PF and plasma RANTES concentrations were significantly elevated in the ovarian cancer patients compared to the group of non-malignant ovarian tumors.

There were no significant differences in the plasma RANTES levels based on tumor stage, grade or histology.

Women with serous cystadenocarcinoma, clear cell carcinoma and endometrioid cystadenocarcinoma had significantly higher PF RANTES levels than patients with undifferentiated carcinoma. Women with clear cell carcinoma and patients with endometrioid cystadenocarcinoma had higher PF RANTES levels than women with mucinous cystadenocarcinoma.

We concluded that RANTES production in the peritoneal cavities of ovarian cancer patients depends on the histological type of the tumor cells.

The study was supported by the Grant KBN NN 407 114036 and KBN NN 407 038537.

Biography

Professor Jan Kotarski since 1999 is the Head of the 1st Department of Oncological Gynaecology and Gynaecology, Medical University of Lublin. He is one of the world's leading experts in gynaecologic oncology. He served as a President of Polish Gynaecological Society from 2006 to 2009. Currently he is a member of New York Academy of Science, European Society of Gynaecologic Endoscopy, European Society of Gynaecologic Oncology, Professor Kotarski's latest interests and research focus on clinical and experimental immunology and immunotherapy of gynaecological malignancies. He is one of the pioneers of dendritic cell vaccination use in the treatment of ovarian cancer.

Hari K Koul, J Cancer Sci Ther, 3:7
doi: http://dx.doi.org/10.4172/1948-5956.S1.11

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Sam-Pointed Domain Ets Transcription Factor-1 (SPDEF-1, a.k.a. PDEF-1) is a Tumor Metastasis Suppressor and its Mechanism(s) of Action

Hari K Koul

University of Colorado School of Medicine, Aurora

Conventional therapies produce a high rate of cure for many patients with cancer, but there is, at present, limited effective treatments for intervention in metastatic cancer. Therefore, at present there is an urgent and unmet need for identifying new targets that could be exploited for intervention in metastatic disease. Progression of cancer from focal to metastatic cancer requires deregulation of growth control, invasiveness and cell motility. SPDEF/PDEF is the latest family member of the ETS transcription factor family, although it is unique in many aspects. PDEF was first discovered as an mRNA transcript highly expressed in prostate tumor cells where it regulates prostate-specific antigen (PSA) gene expression and is an androgen receptor co-regulator. SPDEF/PDEF expression is highly restricted to epithelial cells and has been found in prostate, breast, colon, ovary, stomach, and airway epithelium. Our recent studies demonstrated that SPDEF/PDEF is lost in a graded fashion as prostate cancer cells advance to aggressive stage (Molecular Cancer, 2010). Strong preclinical evidence is emerging that SPDEF/PDEF is a negative regulator of tumor progression and metastasis. PDEF expression is often lost in late-stage, advanced tumors. The induction of tumor aggressiveness in response to the loss of PDEF is thought to be due to the plethora of PDEF-regulated gene targets, many of which are known players in tumor progression including tumor cell invasion and metastasis (Cancer Letters 2011). Specifically our studies point to the direct regulation of MMP-9, a tumor progression associated MMP that is associated with cancer metastasis, by PDEF. These data lead us to the hypothesis that PDEF is a tumor metastasis suppressor protein. Current studies in our lab are aimed at understanding molecular mechanism/s involved in regulation of cancer cell metastasis by SPDEF/PDEF as well as mechanisms of SPDEF/PDEF silencing during cancer progression form indolent to aggressive metastatic phenotype.

Biography

Professor Hari K Koul is the founder Program Director of Urosciences Program and Cell Signaling and Molecular Urology Laboratory; Professor (with Tenure) and Director of Research, Department of Surgery-Division of Urology at The University of Colorado School of Medicine, Anschutz Medical Campus-Aurora-CO-USA; Research Biologist the Department of Veterans Administration Health Center-Denver-CO-USA; Professor Department of Bioengineering, and Program in reproductive Sciences; and Professor/ full member of the Developmental Therapeutics program at The University of Colorado Comprehensive Cancer Center Anschutz Medical Campus-Aurora-CO-USA. He is an internationally recognized researcher and over past two decades, Dr. Koul has greatly contributed to our understanding of molecular mechanisms, specifically signal transduction pathways in genitor-urinary disorders including prostate and bladder cancer. Dr. Koul's research is currently focused in understanding the role of Hypoxia/ re-oxygenation and the resulting ROS in mediating aggressive phenotypes in solid tumors in general and prostate, bladder and kidney cancer in particular. In addition his laboratory is engaged in deciphering the molecular signatures of aggressive renal and prostate tumors; and has recently identified MMP9 as a downstream target of Prostate Derived Ets transcription Factor (PDEF). Dr. Koul has been elected Fellow of the American Society of Nephrology (FASN: since 2004), and A Fellow of the American College of Nutrition (FACN; since 2002). Dr. Koul earned M.Sc. (Biochemistry-1986) from Kashmir University-Srinagar, J&K-India and Ph.D. (Biochemistry-1990) from PGI, Chandigarh, India. As a graduate student Dr. Koul was a recipient of prestigious fellowships from CSIR-India. Dr. Koul came to USA in 1991 on a NIH-post-doctoral fellowship and worked as a post-doctoral fellow (1991-1994) at the University of Massachusetts Medical School, in Worcester, MA. He was promoted to Jr. Faculty position at UMASS Medical School and continued to work there until 1996. Dr. Koul served as Sr. Staff Scientist and founding member of Urology Research team at Henry Ford Health Sciences Center/ Case Western University, Detroit MI from 1996-2003, when Dr. Koul was recruited to head the Urology Research Program at the University of Colorado Denver, School of Medicine.