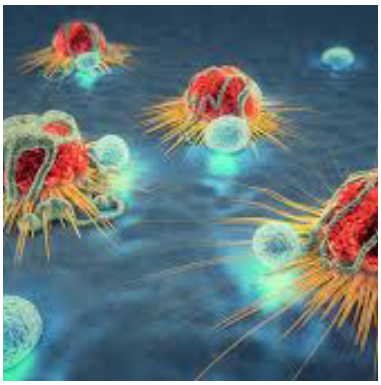
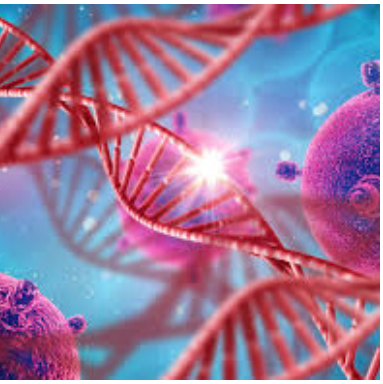
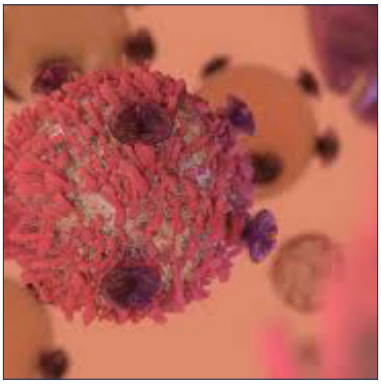
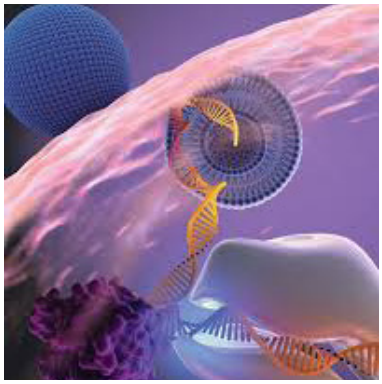
Keynote Forum
March 18-19, 2019

Oncology 2019 & Cancer Therapy 2019



International Conference on

Oncology & Cancer Therapy

March 18-19, 2019 | London, UK

Stanley P L Leong

California Pacific Medical Center and Research Institute, USA

Cancer metastasis from the primary site to the sentinel lymph nodes and beyond in relationship to the immune system

Multiple factors are involved in the development of cancer. They may include carcinogens, host genetic risk, chronic inflammation and others, which cause mutations within cellular DNA. The mutant cancer cells flourish in the cancer microenvironment (CM) consisting of fibroblasts, lipocytes, immune cells, lymphatic and vascular vessels and other parenchymal cells. Mutation gives rise to the unique characteristics of cancer heterogeneity with various clones competing to survive within the CM. By evading the host immune surveillance and by its intrinsic proliferative advantages using unique signaling pathways, cancer clones grow by expansion. The cancer cells tend to spread first through the sentinel lymph node (SLN) in over 90% of the time, which serves as a primary gateway for the cancer cells to proliferate and spread further to distant sites. Patients with negative SLNs but subsequently develop distant metastasis during follow-up indicate that their cancer cells have bypassed the SLNs to spread through the vascular system. VEGF-C has been found to induce lymph angiogenesis in the SLNs and facilitate systemic metastasis. The interaction between cancer cells and the immune system varies among different patients and it undergoes continuous dynamic changes. The relationship between the cytotoxic T cells (CTLs) and cancer cells is highly complex and their molecular interactions have been elucidated through the understanding of the CTLA-4 and programmed death (PD-1) pathways. During cancer progression and evolution in the host, the cancer cells have maximized their ability to take advantage of the CTLA-4 and PD-1 pathways to proliferate while causing the CTLs to undergo apoptosis and wither away so that the cancer cells can grow without hindrance. Thus, aggressive cancer clones have achieved the survival advantage as the 'fittest' clones akin to Darwin's survival of the fittest from the influence of natural selection. The CM may exert the selective force to favor the cancer clones to develop, similar to the principles of directed evolution of enzymes and antibodies The molecular relationship between cancer growth and CM as well as the host influence such as the immune system on cancer progression may be studied by using multiplexed microscopy, genomic profiling, microRNA analysis and gene exon sequencing. To date, blockade of the immune checkpoint pathways such as ipilimumab (anti-CTLA-4), pembrolizumab and nivolumab (both anti-PD-1) have resulted in significant tumor responses with subsequent FDA approval of these drugs. The immune system and cancer growth are so complex that perhaps artificial intelligence needs to be developed to elucidate the proliferation of cancer cells in relationship to the structure and physiology of the lymphatic system in a new field, which may be coined as Oncolymphology.

Speaker Biography

Stanley P L Leong is board certified in surgery and is an internationally recognized surgical oncologist with expertise in melanoma. He specializes in sentinel lymph node surgery and immunotherapy for patients with advanced melanoma. He has lectured nationally and internationally on new advances in the treatment of malignant melanoma and the use of selective sentinel lymphadenectomy. As Associate Director of CPMC's Center for Melanoma Research and Treatment, he collaborates with other investigators including Mohammed Kashani-Sabet on a new integrated research program at CPMCRI aimed at developing novel combination therapies for aggressive and metastatic tumors, including melanomas. He is the founding member and president of the Sentinel Node Oncology Foundation. He has chaired and co-chaired the biennial International Cancer Metastasis Congress since 2005 with emphasis in the mechanisms of cancer metastasis through the lymphovascular system.

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Raghu Pandurangi

Sci-Engi-Medco Solutions, USA

A prior activation of apoptosis pathways of tumor (AAAPT) technology: Biomarker for the risk stratification of cancer patients

Statement of the Problem: Cancer cells desensitize themselves to circumvent interventions. Consequently, apoptosis index (AI, Apoptosis level) gets reduced making them opaque to treatments. Methods are needed to improve the extent response from treatments and to predict which treatment works better for which patients.

Potential Solution: The potential solution would be to a) enhance the cell death selectively in tumor, image cell death in tumor, measure AI using non-invasive imaging technology SPECT, PET, Ultrasound and MRI), b) sensitize low and non-responsive tumors using AAAPT technology and c) use AI as a biomarker to predict the efficacy of treatments.

Results: The leading AAAPT drug molecules sensitized cancer stem cells and low-responsive tumor cells by reducing the IC-50 of several FDA approved drugs (e.g. doxorubicin, paclitaxel, gemcitabine) by 10-15 times in vitro. As a result, the combination of AAAPT with chemotherapy achieved tumor regression in an in vivo xenograft triple negative breast cancer (TNBC) tumor model at a much lower dose of chemotherapy and reduced dose resulted cardiotoxicity. SPECT-CT images showed an increase in apoptosis in tumor selectively (increasing efficacy), while reduced the cell death in heart (reducing cardiotoxicity). The potential mechanism of drug action is depicted in the image attached.

Conclusion and Significance: Imaging spontaneous tumor apoptosis index permitted the risk stratification of patients as to who responds better to which treatments based on tumor AI. The broader significance of AAAPT is that it can, potentially be used as a neoadjuvant to chemotherapy, radiation therapy, immunotherapy or radionuclide therapy and clinically translatable for a better management of cancer patients.

Reference: Raghu Pandurangi: A priori Activation of Apoptosis Pathways of Tumor Technology (AAAPT) for Enhancing Tumor Cell Response to Anticancer Agent, Jan 2016, PCT/US16/68554.

Speaker Biography

Raghu Pandurangi started his scientific career PhD in spectroscopy followed by post-doctoral training at Radiology and Internal medicine, University of Missouri, Columbia where he remained as a faculty for 10 years. He was a principle investigator position in Shering AG, Germany where he directed and involved in 2 FDA approved drugs (AccuTect and NeoTect). He was a team leader at Mallinckrodt directing apoptosis imaging. He became an entrepreneur in 2013 inventing AAAPT technology for improving FDA approved drugs. Currently, he is the Founder, President and CSO of Sci-Engi-Medco Solutions (SEMCO) and Amplexi-LLC, recipient of several NIH grants and awards.

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Notes:

Stefan Glueck

Celgene Corporation, USA

Finally we have positive data in the treatment of breast cancer using ICI

The idea of using the immune system to fight cancer is over 100 years old. (Paul Ehrlich's "Magic Bullet"). A new molecular approach led to a better understanding of the immune system. Check point regulation, understanding roles of Tregs, Th1 and Th2, development of CAR-T cells, as well as regulation of DC and Macrophages, has led to discovery of immune checkpoint inhibitors (ICI) and modulators that are currently used in studies of several STs. Positive studies have led to the US FDA approval of a number of these compounds but none to date are approved in breast cancer (BrCa). Moreover, PD-1 / PDL-1, MSI high (and dMMR), MTB are the currently "best" predictive markers for IO therapy. BrCa actually has some of these markers positive only in subsets and less frequently expressed than most other tumors e.g. malignant melanoma or non-small cell lung cancer and others. In order to improve the potential efficacy of ICI in breast cancer, the addition of chemotherapy was one of the strategies. Many early and large clinical trials in all phases, are underway in BrCa and will be reported in 2018 and 2019. We will discuss the mechanism of action, its impact on BrCa and some of the early results and next generations ICI.

Speaker Biography

Stefan Glueck is the Vice President, Global Medical Affairs, at Celgene Corporation since October 2014, and a medical oncologist with focus on breast cancer. He has overseen oncology activities worldwide, as well as the Immuno-Oncology Program in solid tumors and hematology. Recently, his job requirements have shifted to include Early Assets. He was presented the "America's Top Oncologists" 2008 award from Consumers' Research Council of America, as well as "Best Doctors in America" honor since 2006 and has annually earned that prestige every year to 2014. This award was warranted after less than 3 years of working in the United States. He is a member of such prestigious professional organizations, as the American Society of Clinical Oncology, European Society for Medical Oncology, American & European Association of Cancer Research, and the International Association for Breast Cancer Research. He has authored or co-authored over 270 articles. In addition, he has written or co-written several book chapters and numerous journal abstracts and has presented more than 380 papers at national and international meetings.

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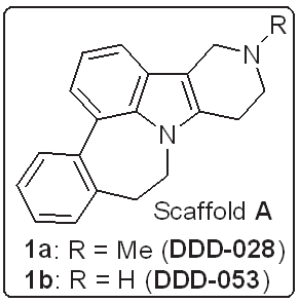
Notes:

Raghavan Rajagopalan

Daya Drug Discoveries, USA

Disease modifying non-opioid analgesic for chemotherapy induced peripheral neuropathy (CIPN)

Chemotherapy induced peripheral neuropathy (CIPN) is debilitating pain condition that results from the use of anticancer drugs such as taxanes, platinum compounds vinca alkaloids, and the like. At present, CIPN is being managed, albeit poorly, with diverse drugs comprising opioids, cannabinoids, anti-epileptics, and antidepressants. Our extensive work on pentacyclic pyridoindole scaffolds resulted in identification of such an analgesic, DDD-028 (1a), for the potential treatment of CIPN. DDD-028 displays potent analgesic activity in paclitaxel induced neuropathy (CIPN). The in vivo study involving chronic administration of paclitaxel along with DDD-028 over an 18-day period demonstrated that DDD-028 is exerting a prophylactic effect against CIPN. Tissue analysis of the spinal cord and the key areas of the brain demonstrates that DDD-028 is preventing the nerve damage by inhibiting glial cell proliferation and changes in morphology. In the receptor binding and selected functional studies, DDD-028 showed no activity at any of the opioid, cannabinoid, or dopamine receptors. DDD-028 is well tolerated in all of the tests and does induce any sedation in any of the animals.



(Fig. 1)

Speaker Biography

Raghavan Rajagopalan is Founder and Chief Scientific Officer of Daya Drug Discoveries Inc., and its affiliate Daya CNS, LLC. He received his PhD in Organic Chemistry from Columbia University in New York, NY, B.S. in Chemistry from State University of New York, Stony Brook, NY, a graduate certificate in Applied Mathematics from Washington University in St. Louis, MO. He did his post-doctoral research in immunochemistry the department of microbiology, Columbia University, New York NY. He is an innovative organic/medicinal chemist and a Registered Patent Agent with the United States Patent & Trademark Office. He has over 35 years of experience in diagnostic and therapeutic drug discovery and developmental research related to four key areas: oncology, neuroscience, nephrology, and infectious diseases. During that time, he has been engaged in small molecules drug discovery in cancer phototherapy, chronic kidney disease, pain, drug addiction, and ADHD. He is principally responsible for the chemistry section for IND and NDA application for 4 drug candidates, 1 of which approved and commercialized with another one undergoing Phase 2 clinical trials. He has over 100 patents and 52 professional publications and presentations of which 3 are landmark publications in kidney disease and cancer phototherapy.

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Notes:

Olivier E Pardo

Imperial College, UK

Targeting RSK4 prevents both chemoresistance and metastasis in lung and bladder cancer: Potential of re-purposed floxacins as novel therapeutic agents

Lung and bladder cancers are mostly incurable due to early development of drug resistance and metastatic dissemination. Hence, novel therapies that tackle these two processes are urgently needed to improve clinical outcome. We have identified RSK4 as a promoter of drug resistance and metastasis in lung and bladder cancer cells and silencing this kinase sensitises to therapy and hinders metastasis in vitro and in vivo. This is mediated through inhibition of the NFkB pathway and the transcription of anti-apoptotic proteins such as BCL2, cIAP1 and cIAP2. Drug screening revealed several floxacin antibiotics as potent RSK4 activation inhibitors and trovafloxacin reproduces all effects of RSK4 silencing in vitro and in vivo. Through crystallography and Markov transient analysis, we propose a mechanism for the action of this compound. Finally, we show that patients undergoing chemotherapy and adhering to prophylactic levofloxacin in the large placebo-controlled randomised phase3 SIGNIFICANT Trial had significantly increased long-term overall survival times. Hence, we suggest that RSK4 inhibition represents a novel therapeutic strategy for treating lung and bladder cancer.

Speaker Biography

Olivier E Pardo graduated from the Faculty of Pharmacy Paris-V, France where he was awarded a Doctorate in Industrial Pharmacy (1997). He completed his PhD at Imperial College-London (2002), UK and subsequently joined the laboratory of Prof. Julian Downward at the CRUK-LRI as a post-doctoral fellow. In 2006, he became team leader at Imperial College-London, Department of Surgery and Cancer where he created the Cellular Regulatory Networks lab. His team focuses on understanding the molecular mechanisms underlying chemo-resistance and metastasis in lung and other cancers. This involves multidisciplinary collaborations with other labs in the UK, France, the US, Canada and China bringing in biochemistry, molecular biology, physics and bio-informatics expertise. The data generated by his lab led to the initiation of several clinical trials in lung and breast cancer patients.

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Notes:

Jan Jacques Michiels

Goodheart Institute, The Netherlands

Change of the 2008/16 WHO into 2018 clinical, laboratory, molecular and pathobiological (WHO-CLMP) criteria for diagnosis of the meloproliferative neoplasms JAK2^{V617F} trilinear polycythemia vera (PV), JAK2 exon 12 PV and JAK2^{V617F}, CALR or MPL^{515} mutated thrombocythemias and secondary myelofibrosis

The JAK2^{V617F} mutated trilinear myeloproliferative neoplasms include a broad spectrum of clinical laboratory and bone marrow features in essential thrombocythemia, prodromal polycythemia vera and erythrocythemic PV, classical PV and advanced stages of masked PV and PV complicated by splenomegaly and secondary myelofibrosis. Heterozygous JAK2^{V617F} mutated ET is associated with low JAK2 allele and MPN disease burden and normal life expectance. In combined heterozygous and homozygous or homozygous JAK2^{V617F} mutated trilinear MPN, the JAK2 mutation load increases from less than 50% in prodromal and early stage PV to above 50% up to 100% in classical PV, advanced PV and PV with MF. Bone marrow histology features show various degrees of diagnostic erythrocytic, megakaryocytic and granulocytic myeloproliferation in JAK2^{V617F} mutated trilinear MPN clearly differ from monolinear megakaryocyticor dual megakaryocytic granulocytic myeloproliferation in MPL or calreticulin mutated thrombocythemia without features of PV. The morphology of clustered large pleomorphic megakaryocytes with hyperlobulated nuclei are similar in JAK2^{V67F} thrombocythemia, prodromal PV and classical PV patients. Monolinear megakaryocytic myeloproliferation of large to giant megakaryocytes with hyperlobulated staghorn like nuclei is the hallmark of MPL^{515} mutated normocellular thrombocythemia. CALR mutated thrombocythemia usually presents with high platelet count around 1000x109/l and normocellular megakaryocytic proliferation of immature megakaryocytes with cloud-like hyperchromatic nuclei followed by dual megakaryocytic granulocytic myeloproliferation followed by various degrees of bone marrow fibrosis. Natural history and life expectancy of MPN patients are related to the response to treatment and the degree of anemia, splenomegaly, myelofibrosis and constitutional symptoms. The acquisition of epigenetic mutations at increasing age on top of MPN disease burden independently predict unfavorable outcome in JAK2^{V617F}, MPL515 and CALR mutated myeloproliferative neoplasms, which mutually exclude each other.

Speaker Biography

Jan Jacques Michiels is a Lifestyle Physician and Medical Doctor, educated in Internal Medicine, Hematology, blood coagulation and Vascular Medicine and graduated as PhD at the Erasmus University Medical Center, Rotterdam. He frequently served as Guest Editor and was the Founder and Editor in Chief of Seminars in Vascular Medicine. He is the Founder of the Thrombocythemia Vera Study Group, the European Working Group on Myeloproliferative Disorders and Myeloproliferative Neoplasms as scientific working groups of the European Hematology Association. He is the founder of the Goodheart Institute & Foundation in Nature Medicine & Health, Rotterdam, The Netherlands.

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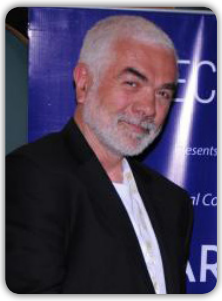

Claude Prigent

Université de Rennes, France

The mitotic kinase Aurora kinase A localises to mitochondria to control organelle dynamics and energy production: Implication for cancer cells overexpressing Aurora-A

Many epithelial cancers show cell cycle dysfunction tightly correlated with the overexpression of the serine/threonine kinase Aurora A (AURKA). Its role in mitotic progression has been extensively characterised, and evidence for new AURKA functions emerges. Here, we reveal that AURKA is located and imported in mitochondria in several human cancer cell lines. Mitochondrial AURKA impacts on two organelle functions: mitochondrial dynamics and energy production. When AURKA is expressed at endogenous levels during interphase, it induces mitochondrial fragmentation independently from RALA. Conversely, AURKA enhances mitochondrial fusion and ATP production when it is over-expressed. We demonstrate that AURKA directly regulates mitochondrial functions and that AURKA over-expression promotes metabolic reprogramming by increasing mitochondrial interconnectivity. Our work paves the way to anti-cancer therapeutics based on the simultaneous targeting of mitochondrial functions and AURKA inhibition.

Speaker Biography

Claude Prigent is a Director of Research CNRS and Head of the Cell Cycle team, IGDR. He has been elected as an Associate Professor at the University Laval, Quebec, Canada. After completing his Post-doc in the DNA repair filed under the direction of Thomas Lindahl at the ICRF in London he has been working on mitosis trying to understand how this cell cycle stage was control by phosphorylation. He focused his activity on the Aurora-A kinase and cancer.

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Notes:

Jan Jacques Michiels

Goodheart Institute, The Netherlands

The PVSG/WHO versus the clinical, laboratory, molecular and pathological (2018 CLMP) defined myeloproliferative neoplasms caused by $JAK2^{V617F}$ $JAK2^{EXON12}$, CALR, MPL and TPO driver mutations are distinct blood & coagulation disorders: Prognostic and therapeutic implications towards 2020 and beyond

The $JAK2^{V617F}$ mutated trilinear myeloproliferative neoplasms (MPN) include a broad spectrum of clinical laboratory and bone marrow features in essential thrombocythemia (ET), prodromal polycythemia vera (PV) and erythrocythemic PV, classical PV and advanced stages of masked PV and PV complicated by splenomegaly and secondary myelofibrosis (MF). Heterozygous $JAK2^{V617F}$ mutated ET is associated with low JAK2 allele and MPN disease burden and normal life expectance. In combined heterozygous and homozygous or homozygous $JAK2^{V617F}$ mutated trilinear MPN, the JAK2 mutation load increases from less than 50% in prodromal and early stage PV to above 50% up to 100%in classical PV, advanced PV and PV with MF. Bone marrow histology features show various degrees of diagnostic erythrocytic, megakaryocytic and granulocytic (EMG) myeloproliferation in $JAK2^{V617F}$ mutated trilinear MPN clearly differ from monolinear megakaryocytic (M) in MPL or dual megakaryocytic granulocytic (MG) myeloproliferation in calreticulin (CALR) mutated thrombocythemia without features of PV. The morphology of clustered large pleomorphic megakaryocytes with hyperlobulated nuclei are similar in JAK2V67F thrombocythemia, prodromal PV and classical PV patients. Monolinear megakaryocytic (M) myeloproliferation of large to giant megakaryocytes with hyperlobulated staghorn like nuclei is the hallmark of MPL515 mutated normocellular thrombocythemia. CALR mutated thrombocythemia usually presents with high platelet count around $1000x10^9$/l and normocellular megakaryocytic (M) proliferation of immature megakaryocytes with cloud-like hyperchromatic nuclei or prefibrotic dual megakaryocytic granulocytic (MG) myeloproliferation followed by various degrees of bone marrow fibrosis. Natural history and life expectancy of MPN patients are related to the response to treatment and the degree of anemia, splenomegaly, myelofibrosis and constitutional symptoms. The acquisition of epigenetic mutations at increasing age on top of MPN disease burden independently predict unfavorable outcome in $JAK2^{V617F}$, MPL^{515} and CALR mutated MPNs, which mutually exclude each other. Current treatment options in MPN include low dose aspirin in JAK2 and MPL mutated ET, phlebotomy on top of aspirin in PV, pegylated interferon in intermediate stages of PV and CALR and MPL mutated ET followed by hydroxyurea and or ruxolitinib in the hypercellular stages of PV and MF.

Speaker Biography

Jan Jacques Michiels is a Lifestyle Physician and Medical Doctor, MD, educated in Internal Medicine, Hematology, Bloodcoagulation and Vascular Medicine and graduated as PhD at the Erasmus University Medical Center, Rotterdam. He frequently served as Guest Editor and was the Founder and Editor in Chief of Seminars in Vascular Medicine. He is the Founder of the Thrombocythemia Vera Study Group, the European Working Group on Myeloproliferative Disorders and Myeloproliferative Neoplasms as scientific working groups of the European Hematology Association. He is the founder of the Goodheart Institute & Foundation in Nature Medicine & Health, Rotterdam, The Netherlands.

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Elena Drozdova

Samara State Medical University, Russia

Cancer and peripheral deep vein thrombosis are both independent diseases: Causes, pathogenesis, conclusions

There is a thesis: CANCER has an often COMPLICATION such as VEIN THROMBOSIS. Cancer is the second leading cause of death globally and is responsible for an estimated 9.6 million deaths in 2018 (The World Health Organization). Sickness rate of deep vein thromboses is approximately 100 per 100 000 population annually. However, having worked as a vascular surgeon for several years I was wondering, why I have never detected cancer? So I decided to analyze 100 cases of morbidity of deep vein thromboses. Thus, for the period 2011-2018 there were identified 3 cases of cancer out of 100 cases of deep vein thromboses. Taking into account a rarity of cancer detection within the group of patients with deep thromboses, I decided to select a control group of 100 people with cancer who were hospitalized for the planned and urgent surgery to find out whether they had ever had deep vein thromboses, any deviations in their coagulograms or whether they had some current problems with the post-thrombotic complications.

Speaker Biography

Elena Drozdova a cardiovascular surgeon. Having taken an extensive practice in the field of vascular surgery, she mainly specializes in the problems of vein thromboses, urgent vascular surgery and vascular access for the patients on hemodialysis. She graduated from Samara State Medical University in 2006. In 2007 after the specialization in Samara Regional Clinical Cancer Center she got a certificate in General Surgery. Later in 2012 she finished Cardiovascular Surgery Residency at Samara State Medical University and got a Certificate of Cardiovascular Surgery. She is an author of a number of medical articles and was offered the position of editorial board member in some journals. Research interest: There are some inconsistencies between practice and dogmas; moreover, she thinks that many of laboratory tests make it more difficult to analyze the data because of their number and low specificity, so we can see neither solution nor direction in which this problem may be solved.

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Kazuko Tatsumura

Gaia Holistic Health, USA

Effects of far-infrared & terahertz Onnetsu therapy on various cancers, rheumatoid arthritis and other diseases

Introduction: Onnetsu means comfortable heat. Onnetsu Therapy invented by Dr. Kazuko Tatsumura emits from a special patented ceramic; 1) Heat 2) Precise 8-10μ of vibration of Far Infrared SunRay and 3) Vibration of Terahertz.

Methods: When Onnetsuki is slid over the skin, healthy areas are comfortable, but IF deep tissue is unhealthy or cold, degenerated, patient feels this spot to be 'hot'. When this 'hot spot' is effectively treated with Onnetsu Therapy (Far-Infrared & Terahertz vibrations, and Heat), the hot sensation subsides and the Disease Conditions improve through vibrating water molecules of our deep tissue. Therefore, the Onnetsu Therapy is both a diagnostic and therapeutic.

Dr. Kazuko's Onnetsu Therapy is based on four historical and scientific facts.

1. Traditional Japanese Concept of the significance of Body Temperature. Hippocrates also has left quotes on Heat.
2. NASA's finding regarding Far-Infrared vibration from Sun light precise 8-10μ. Also, added is the specific Terahertz vibration of earth minerals from volcanos stones from the depth of our planet earth.
3. Immunology by Dr. Toru Abo, balancing autonomic nervous system to improve condition of white cells; Raising Immunity.
4. Promoting four flows of Energy throughout our body by using acupuncture meridian technique.

Result: Some countries (Peru, Cuba & Mexico) are practicing it in the hospitals and clinics. Clinical trials have shown improvements on many diseases: such as asthma, brain, ear & eye problems, cancers, diabetes, rheumatoid arthritis, tuberculosis and various pain conditions.

Conclusion: Onnetsu Therapy is a new, easy & noninvasive treatment modality to treat difficult chronic medical conditions. Therapy uses Universal Vibrations, Heat, Light, Autonomic Nervous System Balance and Acupuncture Meridian System.

Speaker Biography

Kazuko Tatsumura graduated from Toho Academy of Music in Tokyo, as a pianist and composer, invited by the Boston Symphony. She then received Master of Art from New York University and finished her PhD credits in Philosophy in 1965.She studied Oriental Traditional Medicine of Japan, Korea, Taiwan and China. In 2000, she received her PHD and OMD from the International Academy of Education in Tokyo. She established the Oki-Do Holistic Health Center in 1994 in NY and in 2001 the GAIA Holistic Center (501C3 nonprofit organization). She invented special holistic healing method called ONNETSU THERAPY using Heat and 2 vibrations of Sun Far Infra-red (8-10μ) and Terahertz from deep earth planet minerals. The therapy has been widely hailed in hospitals and clinics especially in South Americas. Her invention is patent pending worldwide.

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