magnus Group

5TH EDITION OF INTERNATIONAL

CANCER CONFERENCE

SEPT 16-17

VIRTUAL EVENT

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BOOK OF ABSTRACTS

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INTERNATIONAL CANCER CONFERENCE

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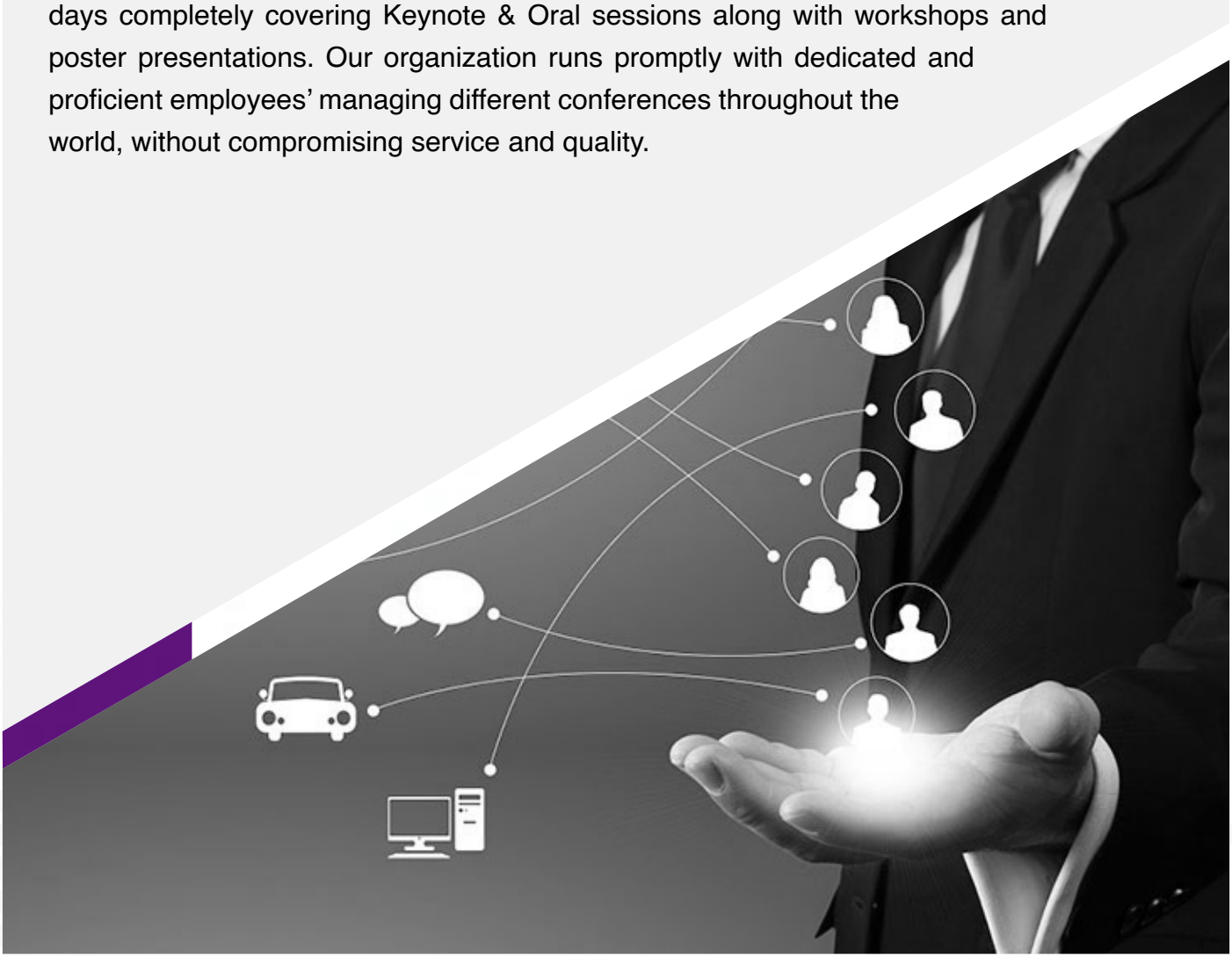
ABOUT MAGNUS GROUP

Magnus Group (MG) is initiated to meet a need and to pursue collective goals of the scientific community specifically focusing in the field of Sciences, Engineering and technology to endorse exchanging of the ideas & knowledge which facilitate the collaboration between the scientists, academicians and researchers of same field or interdisciplinary research. Magnus group is proficient in organizing conferences, meetings, seminars and workshops with the ingenious and peerless speakers throughout the world providing you and your organization with broad range of networking opportunities to globalize your research and create your own identity. Our conference and workshops can be well titled as 'ocean of knowledge' where you can sail your boat and pick the pearls, leading the way for innovative research and strategies empowering the strength by overwhelming the complications associated with in the respective fields.

Participation from 90 different countries and 1090 different Universities have contributed to the success of our conferences. Our first International Conference was organized on Oncology and Radiology (ICOR) in Dubai, UAE. Our conferences usually run for 2-3 days completely covering Keynote & Oral sessions along with workshops and poster presentations. Our organization runs promptly with dedicated and proficient employees' managing different conferences throughout the world, without compromising service and quality.



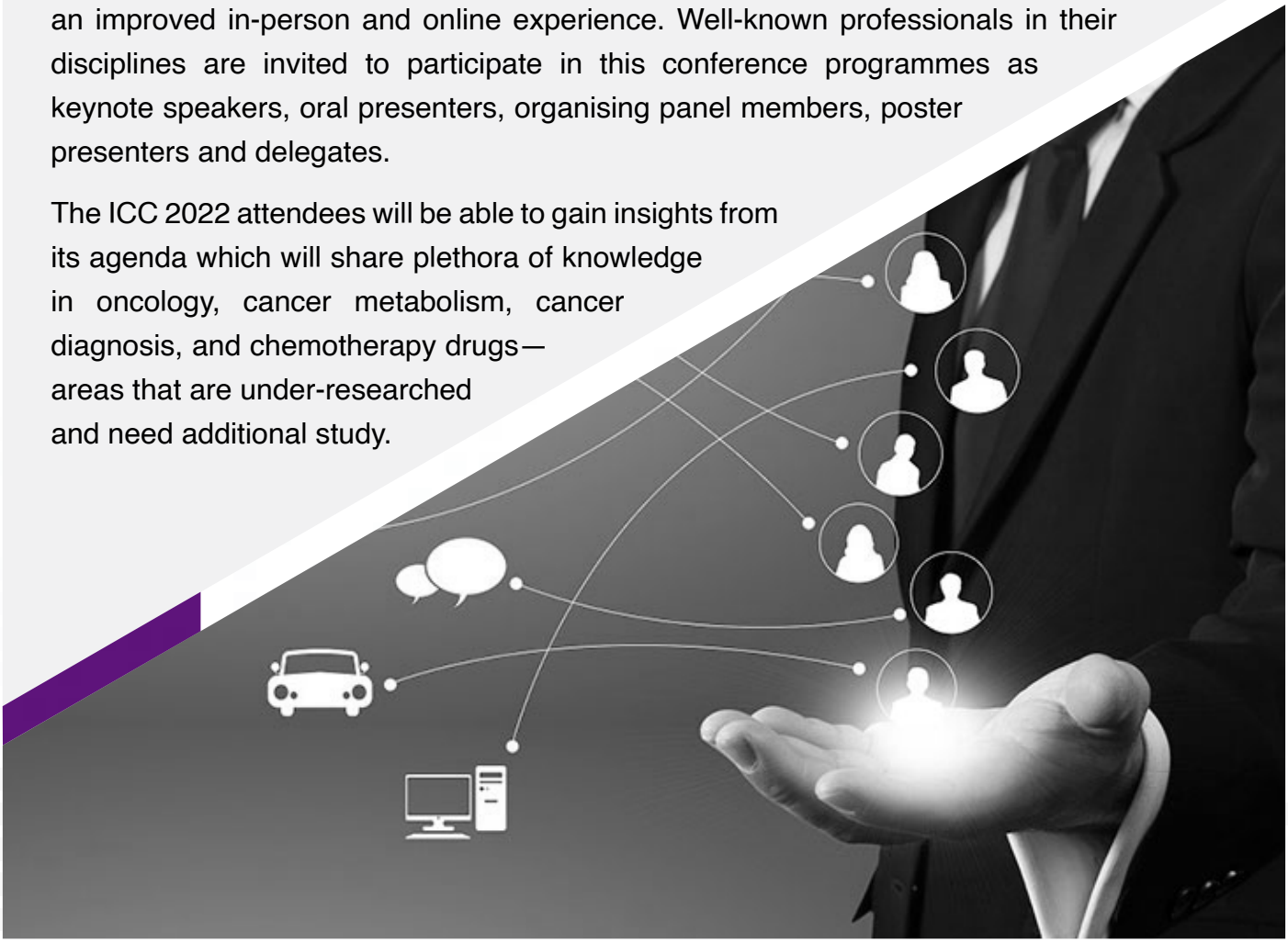
ABOUT ICC 2022

Since 2016, we have been bringing together experts in the field of cancer at our conferences. We are ecstatic to announce the upcoming event in this series ICC 2022 which will be taking place virtually during the dates September 16-17, 2022. The “5th Edition of International Cancer Conference” will be driven by the theme “Stretching Wings to Fight Cancer through Unified Research Approaches.”

The ICC congress is a one stop solution for oncology gathering and is recognised widely as a forum for professionals from the oncology and cancer research community worldwide, including doctors, scientists, researchers, patient activists, caregivers, journalists, pharmacists, oncologists, cancer experts, and industry representatives. The global symposium will provide a hybrid platform for interaction, communication, and education on oncology related topics which will give attendees the rare opportunity to share the valuable work that balances clinical, translational, and basic research.

The summit will showcase the most recent developments in cancer therapy and provide a strong educational programme, as well as possibilities for delegate interaction through an improved in-person and online experience. Well-known professionals in their disciplines are invited to participate in this conference programmes as keynote speakers, oral presenters, organising panel members, poster presenters and delegates.

The ICC 2022 attendees will be able to gain insights from its agenda which will share plethora of knowledge in oncology, cancer metabolism, cancer diagnosis, and chemotherapy drugs—areas that are under-researched and need additional study.



KEYNOTE FORUM

DAY 01

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Veronica J. James

Professor of Science, Australian National University, Australia

Low angle X-ray fibre diffraction wins gold for Cancer diagnosis

Every cancer sends o*ut* a type-specific signal, as soon as it starts, thus providing the earliest possible diagnosis of the cancer by the cancer itself. These signals can be detected as specific alterations in the low angle X-ray diffraction patterns for skin and/or hair. In addition for some cancers, the intensity of such a signal can also indicate the grade of the cancer. Some such cancers are breast cancer and colon cancers. For other cancers, such as prostate cancers and melanoma the grade of the cancer is indicated by the width-range of the diameter of the added ring. In the case of prostate cancer, the ring moves from between the 13^{th} and 14^{th} orders for Gleason 5 to between the 13^{th} and 15^{th} order for Gleason7 and past the 15^{th} for higher Gleason scores. More rings are involved for other cancers. If more than one cancer is present, each will give its own signal. All signals disappear if and only if the cancer is successfully removed, thus confirming the total removal of the cancer by surgery and/or chemotherapy. Since the added rings in the skin or hair patterns are provided by the cancer itself, these tests must always be one hundred per cent accurate, no further verification is necessary. Over four thousand tests have been carried out for breast cancer. Seven hundred and sixty from Finland. Six false positives were found, all of which subsequently were found not to be false. A sensitivity of 100% was recorded for this test and tests for all cancers that have been studied to date.

Biography

Veronica J James completed her PhD in Physics from the University of NSW in 1971. Working in crystallography, she published 40 papers on the molecular structures of small organic crystals, before moving into the fibre diffraction studies of collagen and keratin. In this area she has carried out the diffraction study that produced the successful structure for hard α-keratin and also pioneered the fibre diffraction diagnostic tests for breast, colon, prostate cancers and for Alzheimer's Disease. She was awarded an OAM for her Phones for the Deaf Program and her Advanced Physics Programs in 1996.

Michael Thompson*,
Soha Ahmadi and Brian De La Franier
Department of Chemistry, University of Toronto, Canada

Electrochemical point-of-care sensor for early diagnosis of Ovarian Cancer

Ovarian cancer (OC), the most lethal gynecological cancer, globally causes 150,000 deaths, annually. Notably, the 5-year survival rate is generally accepted to be over 90% if the disease is detected at Stage 1. The well-known current CA-125 diagnostic test for OC has been shown to yield both false positive and negative results, and is not normally employed to detect the disease at an early Stage. Accordingly, there is a prescient requirement for the introduction of a low-cost screening test that is rapid, sensitive and selective, and that can be applied on a large-scale basis. In this regard, we have developed a simple, precise, and low-cost screening test using an electrochemical technique to fabricate a point-of-care testing (POCT) device for early detection of OC. The device detects lysophosphatidic acid (LPA), a highly promising biomarker, which has been observed to be elevated in 90% of stage I OC patients, and gradually increases as the disease progresses to later stages. This is achieved via a recognition probe, gelsolin, immobilized on medical-grade stainless-steel electrodes. The detection technology is based on electrochemical impedance spectroscopy (EIS), whereby three-electrode setup is employed which consists of a working electrode, counter electrode, and reference electrode. The working electrode is modified to provide the recognition surface. The main challenge in developing such a tool is overcoming the ubiquitous, problematic phenomenon known as non-specific adsorption (NSA), which is due to the fouling of non-target molecules in target biological fluid on the recognition surface of the device. To overcome NSA, we have used a proprietary strategy using silane-based interfacial chemistry to modify the stainless-steel electrodes. Research on the technology to date has demonstrated that LPA can be detected in serum at the micromolar level. In collaboration with Princess Margaret Hospital, Toronto tests on patient samples are planned

Audience Take Away

- Introduction of a new electrochemical biosensor for assay of a cancer biomarker.
- Detection of lysophosphatidic acid, a marker for ovarian cancer, in serum, addressing the pressing need for an early-Stage detection strategy.
- Proving a solution to the prevalent fouling or non-specific adsorption usually observed for biosensor-based methods.

Biography

Professor Michael Thompson obtained his undergraduate degree from the University of Wales, UK and his PhD in analytical chemistry from McMaster University. Following a period as Science Research Council PDF at Swansea University he was appointed Lecturer in Instrumental Analysis at Loughborough University, UK. He then moved to the University of Toronto where he is now Professor of Bioanalytical Chemistry. He has held a number of distinguished research posts including the Leverhulme Fellowship at the University of Durham and the Science Foundation Ireland E.T.S Walton Research Fellowship at the Tyndall National Institute, Cork City. Thompson has served on the Editorial Boards of a number of major international journals including Analytical Chemistry and The Analyst and is currently Editor-in-Chief of the monograph series “Detection Science” for the Royal Society of Chemistry, UK and Associate Editor of IEEE Sensors. He has been awarded many prestigious international prizes for his research including The Robert Boyle Gold Medal of the Royal Society of Chemistry, The Elsevier Prize in Biosensor and Bioelectronic Technology and the E.W.R. Steacie Award of the Chemical Society of Canada.

Atif A. Ahmed[1]*, Midhat Farooqi[1], Maria Tsokos[2]

[1]Department of Pathology, Seattle Children's Hospital, Seattle, Washington, USA

[2]Department of Internal Medicine, Beth Israel Deaconess Medical Center, Boston, Massachusetts, USA

Digital profiling of protein and microRNA in Rhabdomyosarcoma

Rhabdomyosarcoma exhibits a complex prognostic algorithm based on histologic, biologic and clinical parameters. In contrast to alveolar rhabdomyosarcoma, embryonal (ERMS) and spindle cell and sclerosing rhabdomyosarcoma (SRMS) histologic types exhibit more heterogenous molecular features and variable clinical behavior. The expression of signaling pathways and microRNA sets has not been previously studied in relation to prognosis and pathologic types of rhabdomyosarcoma. To identify relevant biomarkers using NanoString digital technology, we have studied 12 ERMS and SRMS cases with clinical information, categorized into adverse prognosis (n=5) and favorable prognosis groups (n=7) based on differences in patients' overall survival. Formalin-fixed paraffin-embedded tumor tissue was subjected to digital spatial profiling and microRNA analysis. Digital spatial profiling revealed enrichment for PI3K/AKT, MAPK, and apoptosis signaling pathways with over-expression of several pathway members. Compared to the group with favorable prognosis, the adverse prognosis tumors had significantly increased expression of INPP4B ($p<0.05$), that was confirmed with traditional immunohistochemistry. Significant upregulation of miR-3144-3p, miR-612, miR-302d-3p, miR-421, miR-548y and miR-548ar-5p ($p<0.05$) was also noted in the unfavorable prognosis group. In conclusion, NanoString digital molecular profiling is a novel way of deciphering dysregulated signaling pathways and microRNA profiles from formalin-fixed paraffin embedded tumor tissue. Digital profiling may identify prognostic biomarkers in rhabdomyosarcoma.

Audience Take Away

- The audience will understand the importance of identifying prognostic biologic markers in rhabdomyosarcomas.
- The audience will gain insight into the use of modern technology in cancer research. Other faculty could use the results and methods to expand their research and teaching. The method employed in this research will provide a practical solution to the problem of scant tissue specimens in pediatric cancer research. It will augment findings and improve the accuracy of basic in vitro studies.

Biography

Atif A. Ahmed is a senior pediatric pathologist at Seattle Children's Hospital, Seattle, Washington, USA. Dr. Ahmed graduated from Medical School in 1988 and has been in academic practice for more than 19 years. Research interests include pediatric tumor biology and targeted biomarkers in cancer. Dr. Ahmed published more than 100 publications incldluing peer-reviewed articles, book chapters and meeting abstracts. He is the book editor of "Gastrointestinal Stromal Tumors in Adults and Children" and "Anatomic and Clinical Pathology Board Review". He is a member of several professional societies and serves on the editorial board of many high impact journals.

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SPEAKERS

DAY 01

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Giuseppe Ercolano[1]*, Pedro Romero[2], Angela Ianaro[1], Sara Trabanelli[3], Camilla Jandus[3]

[1]Department of Pharmacy, University of Naples Federico II, Naples, Italy

[2]Department of Oncology UNIL CHUV, University of Lausanne, Lausanne, Switzerland

[3]Department of Pathology and Immunology, Faculty of Medicine, University of Geneva, Geneva, Switzerland

Immunosuppressive mediators impair pro-inflammatory innate lymphoid cell function in human malignant Melanoma

Innate lymphoid cells (ILC) are a family of immune cells that are emerging as potent orchestrators of immune responses. In cancer, ILCs display both pro- and antitumorigenic functions depending on the nature of the tumor and the involved ILC subset. Little is known about the ILC–tumor cross-talk in human melanoma. Here, we showed that ILC1s were enriched but functionally impaired in cytokine secretion in both peripheral blood mononuclear cells and tumor-infiltrated lymph nodes of melanoma patients. These findings were confirmed in vivo in murine cutaneous melanoma. Multiple immunosuppressive mechanisms are described in the melanoma microenvironment. Among others, adenosine and kynurenines were shown to suppress antitumor immune responses. By exposing ILCs to adenosine and kynurenines, we observed a similar shift toward the ILC1 subset distribution and impairment in proinflammatory cytokine production to that of patient samples studied ex vivo. Thus, we hypothesized that the immunosuppressive microenvironment of malignant melanoma might shape ILC subpopulations. Hence, we provide a rationale for the use of drugs targeting adenosine and kynurenine pathways in melanoma patients.

Audience Take Away

- In this research, we observed that ILC1s were enriched, but functionally impaired in both peripheral blood mononuclear cells (PBMC) and tumor-infiltrated lymph nodes (TILN) in melanoma patients. These findings were confirmed by using an *in vivo* model of cutaneous melanoma. In addition, we demonstrated that the immunosuppressive metabolites ADO and kynurenines directly modulated the ILC subset distribution and impaired their proinflammatory cytokine secretion, which might have direct implications for the optimization of current immunotherapy strategies. Thus, in the current milieu of promising preclinical and clinical approaches targeting the IDO and adenosinergic immunosuppressive axes, we have generated new evidences to support blocking these pathways in melanoma patients.

Biography

Dr. Giuseppe Ercolano graduated as Pharmacologist in 2014 at the University of Naples Federico II. In 2015, he started his PhD in Pharmaceutical science, at the Department of Pharmacy in Naples. In 2017, he spent one year at the Ludwig Institute for Cancer Research of the University of Lausanne (Switzerland) under the supervision of Prof. Pedro Romero. From 2019 to 2020 he worked as post-doctoral fellow at the University of Lausanne and the Department of Pathology and Immunology of the University of Geneva (Switzerland) under the supervision of Prof. Camilla Jandus. After one year as post-doctoral fellow at the Department of Pharmacy of the University of Naples Federico II (Italy), in 2022, he obtained a position as Researcher (RTDA). To date, he has published 25 papers in high-impact journals.

Jian Kang[1,2]*, Keefe T. Chan[1,2], Xinran Huang[1,2], Natalie Brajanovski[1], Eric P. Kusnadi[1], Anna S. Trigos[1,2], Elaine Sanij[1,2,3], Richard B Pearson[1,2]

[1]Division of Cancer Research, Peter Mac Callum Cancer Centre, Australia

[2]Sir Peter MacCallum Department of Oncology, University of Melbourne, Australia

[3]St Vincent's Institute of Medical Research, Australia

Targeting the ribosomes to treat Blood Cancers

The majority of human tumors are characterized by deregulated signaling through the PI3K/RAS/MYC oncogenic network, leading to an increased rate of ribosome synthesis, mRNA translation and hence protein synthesis. Direct targeting of elevated ribosome biogenesis is becoming a new treatment option for oncogene-driven cancers. We developed the "first-in-class" selective inhibitor of ribosome biogenesis, CX-5461 which inhibits RNA polymerase I transcription of ribosomal RNA (rRNA) genes, the rate-limiting step in ribosome biogenesis. We have demonstrated the remarkable potency of targeting ribosome biogenesis in mouse models of blood cancer. Our first-in-human Phase I trial of CX-5461 in blood cancers demonstrated on-target activity in patients' samples and a signal for anti-tumor activity in a third of the trial's participants (6/ 17 patients). CX-5461-mediated alterations in rRNA synthesis and rDNA chromatin and topology subsequently activate two major signaling pathways including p53-dependent nucleolar stress response and a p53-independent nucleolar DNA damage response involving the activation of ATM/ATR kinase. We further demonstrated a marked improvement in the efficacy of CX-5461 in combination with PI3K/AKT/mTORC1 pathway inhibitors in a Myc-driven B-lymphoma mouse model. We show that this improved efficacy is associated with selectively suppression of translation of mRNAs encoding regulators of cellular metabolism. This exciting finding suggests that specific targeting of mRNA translation is a common mode of action of ribosome-targeting therapies for oncogene-driven cancers. Acquired resistance to this co-treatment is driven by translational re-wiring that results in dysregulated cellular metabolism and induction of a cAMP-dependent pathway critical for the survival of blood cancers including lymphoma and acute myeloid leukemia. We also developed a CX-5461 resistant human AML cell model to characterize the molecular changes associated with CX-5461 response using RNA-seq, proteomics, and phosphoproteomics. Integrative analysis of the transcriptomic and proteomic data revealed key mediators and pathways involved in the response and resistance to CX-5461 in AML cells. Targeting aurora kinase A potentiates CX-5461's cytotoxicity in AML cell models. All these findings provide novel understanding and therapeutic approaches to accelerate this new paradigm into the standard-of-care treatment for patients with blood cancer.

Audience Take Away

- The audience will gain a better understanding of a new treatment option for blood cancers by targeting ribosome biogenesis and mRNA translation. This presentation will dissect the molecular mechanisms of sensitivity and resistance to ribosome-targeting therapy and explore new combination therapies to improve the efficacy in blood cancers. The research models, experimental approaches and strategies can be exploited for mechanistic investigation of other cancer therapies.

Biography

Dr Kang received her PhD degree in molecular medicine at the University of Auckland in 2011. She worked as a postdoctoral researcher at the Garvan Medical Research Institute, Sydney during 2011-2013. In 2013 she joined the research group of Prof. Rick Pearson at the Peter MacCallum Cancer Centre (Peter Mac), Melbourne to develop, characterize and optimize ribosome-targeting therapy. She is a Senior Research Officer at Peter Mac. She has published 29 research articles and made fundamental discoveries on oncogenic signaling transduction, ribosome biogenesis and mRNA translation in cancer.

Haiying Bao [1]*, Zhijun Li[2]

[1]College of Chinese Medicine Materials, Jilin Agricultural University, Changchun, Jilin, China

[2]College of Chinese Medicine Materials, Jilin Agricultural University, Changchun, Jilin, China

The research on anti-tumor activity and its mechanism of traditional chinese medicine *Inonotus hispidus*

The mushroom Inonotus hispidus is traditional Chinese medicine, which has been used to treat tumor illness for a long history in China. I. hispidus mushroom mainly grows on five different tree species (Morus alba L., Ulmus macrocarpa Hance, Fraxinus mandshurica Rupr., Ziziphus jujuba Mill., and Malus pumila Mill.), and their fruiting bodies were all separately used in the market. To discuss the anti-tumor mechanism of I. hispidus petroleum ether extract (IPE) on H22 tumor-bearing mice from the point of view of metabonomics. We found that I. hispidus petroleum ether extract (IPE) has significant anti-tumor activity. Moreover, we explored the potential antitumor regulatory pathways and targets of I. hispidus, and performed whole transcriptome and proteome analyses in the H22 tumor-bearing mice model.The combined omics analysis revealed five key genes/proteins, including Lilrb4a, Nrp1, Gzma, Gstt1 and Pdk4, which may play a role in the anti-tumor pathway and were validated.

Audience Take Away

- To understand the effective mechanism of Chinese traditional medicine *Inonotus hispidus* in anti-tumor and cancer prevention.
- To understand the application prospect of anti-tumor activity of traditional Chinese medicine *Inonotus hispidus.*
- To promote other scholars to join in the study of anti-tumor activity of traditional Chinese medicine.

Biography

Dr. Bao working in the College of Chinese Materia Medica, Jilin Agricultural University. From 1993 to 1996, she studied for a master's degree in Changchun College of Traditional Chinese Medicine(Chinese materia medica), get a Master of Medicine. From 1998 to 2001, she studied for doctor's degree in Jilin Agricultural University(The direction of medicinal fungi), earn a doctorate in agronomy, exceptional promotion to professor in 2003. From 2004 to 2005 Postdoctoral fellow at the School of Pharmacy, Chungnam National University, South Korea(Natural Medicinal Chemistry and Molecular Pharmacology).

Xiaofeng Liu[1]*, Xiaojuan Du[2], Baocai Xing[3]

[1]Hepatopancreatobiliary Surgery Department I, Peking University Cancer Hospital & Institute, China

[2]Department of Cell Biology, School of Basic Medical Sciences, Peking University Health Science Center,China

[3]Hepatopancreatobiliary Surgery Department I, Peking University Cancer Hospital & Institute, China

Targeting HTP1 ameliorates chemoresistance of Colorectal Cancer

Cancer stem cell (CSC)-related chemoresistance frequently causes poor prognosis of the patients with colorectal cancer (CRC). The discovery of the chemoresistance-relevant molecules and elucidation of the underlying mechanisms will provide potential therapeutic clues for CRC. In this presentation, we reported the role of HTP1 in CSC-related chemoresistance of CRC. We identified HTP1 as a potential molecule functioning in CRC chemoresistance by comparing the expression profiles between chemo-resistant and chemo-sensitive CRC tissues. Further functional studies using CRC cell lines and CDX (CRC cell derived xenograft model) demonstrated that HTP1 enhances chemoresistance of CRC by promoting stemness of CRC cells. Moreover, inhibition of HTP1 ameliorates chemoresistance of CRC cells. Mechanistically, we found that HTP1 contributes to upregulate wnt/β- catenin signalling and further increase expression of stemness-related genes. In clinics, the upregulation of CRC is associated with poor outcome of CRC patients. Together, our findings reveal the roles of HTP1 in CRC chemoresistance. Targeting inhibition of HTP1 function might be a potential therapeutic strategy to ameliorate chemoresistance in CRC.

Audience Take Away

- Our findings suggest the roles of HTP1 in CRC chemo-resistance, which is useful and helpful for new drug design.
- Our results expand the understanding about HTP1-mediated CRC progression. This provides new information for researchers in the related field.
- The methods we used are useful and helpful for other scientists in this field.
- We hope to have new collaborations with researchers who are interested in our studies.

Biography

Dr. Xiaofeng Liu studied at Peking University and graduated as Ph.D. in 2017. He then joined the research group of Prof. Xing at Peking University Cancer Hospital & Institute. After two-year postdoctoral fellowship, he obtained the position of an Associate Professor at PUCHI. The research in his laboratory is aimed at developing the potential targets and applying them to improve strategy for treating human CRCs. They employ diverse methods including CRISPR/Cas9-mediated genome editing, High-throughput sequencing, proteomics, and traditional biochemical methods. He has published more than 20 research articles in SCI (E) journals in recent years.

Timothy Makumbi[1]*, Henry Wabinga[2], Moses Joloba[3], Moses Galukande[1], Annettee Nakimuli[4]

[1]Department of Surgery, Makerere University, Kampala, Uganda
[2]Department of Pathology, Makerere University, Kampala, Uganda
[3]Molecular Division of Biomedical Sciences, Makerere University, Kampala, Uganda
[4]Department of Obstetrics and Gynaecology, Makerere University, Kampala, Uganda

Feasibility of micro RNA's profiling among Breast Cancer tissues at a sub Saharan university teaching hospital facility, Uganda.

The incidence of this early onset breast cancer in Uganda has tripled if compared to levels of 1961.Breast cancer in these women is peculiar in its progression and aggressiveness yet hardly an answer has been offered for those characteristics. The ray of hope has emerged through genomic studies of miRNAs in cancer bio-pathologic features. Breast cancer continues to torment and devastate women worldwide. Globally, breast cancer is a leading cause of cancer –related death. among the women population. Cancer of the breast exhibits an unusual aggressive growth pattern among the young population of sub Saharan Africans. Disparities in cancer characteristics are noted between high income and low middle income woman populations.

Carcinoma of the breast is normally a disease of women who have surpassed the reproductive period. However, a peculiar category occurs before the fifth decade of life, irrespective of ethnic, religious, social status, education nor geographical preponderance, termed early onset breast cancer.

Worldwide, studies in response to cancer risk heterogeneity have unveiled a new concept of Micro RNAs in the development of various cancers. MicroRNAs are tiny short single strands of non-coding RNA comprising 16 – 20 nucleotides in length. Expression of MicroRNAs often times manifest as excessive or under production hence termed dysregulation or aberrant expression. MicroRNAs though non protein forming in character (non-coding), possess an essential regulatory role over all protein-producing DNA mediated cellular processes.

There is paucity of knowledge on the prevalent dysregulated MicroRNAs, their impact on disease characteristics and survival of early onset breast cancer in Uganda. Therefore, the overall objective of this study was to evaluate the feasibility of profiling dysregulated miRNAs and describe the role of these small non coding RNAs in early onset breast cancer pathogenesis, bio-pathologic disease characterization and survival at sub Saharan Africa University Teaching Hospital.

Methods: The study site was carried out the Breast and Endocrine unit in conjunction with the accessible Laboratories at the University Medical teaching facility, Mulago hospital in Uganda. The proposed study was meant to be the needs assessment evaluation before delving in a three pronged doctoral study to address for the respective objectives in form of three multi-designed sub-studies.

Sub-study (i) will be a cross sectional design to determine the prevalence and associated factors of dysregulated MiRNAs expression, in objective (i).

Sub-study (ii) will be cross-sectional – Both descriptive and analytical in order to enable the correlation of dysregulated MiRNAs with clinical and pathological tumor characteristics in objective (ii). Sub-study (iii) will be a retrospective cohort study to enable establishment of any association between dysregulated MiRNAs and survival of EOBC.

Results: The needs assessment evaluated the Breast and Endocrine Unit registry for breast cancer. The reviewed and complete patients' records were subjected to random sampling until a sample size of 100 was realized, however if the respective archived block appears overtly poor stored, that record set was excluded from this study. Patients' records and laboratory specimens (fresh and archived formalin fixed histological breast blocks) were assembled to be studied for presence of aberrantly expressed miRNAs. Through a qualitative approach, a sample size of 12 key informants was purposely selected by Canon ball method for this study.

Data were gathered by the Delphi method through short 20 minutes' interviews conducted by the principal investigator. The results were subjected to tailored analyses involving descriptive statistics, frequencies, and the thematic qualitative analyses for meaningful deductions. The majority of the participants agreed that research was addressing a pertinent issue affecting women health worldwide. However, the majority (80%) noted that the feasibility was likely to be impacted by human resource capacity at our laboratories; lack of expertise since not a routinely practiced procedure and escalated cost implications for the tests done locally. Fifty-seven percent, 57% of the participants dis agreed in entirety on the feasibility of this study if it were to be done locally. Forty percent, (40%) though agreed that with an international collaboration, the study is will be feasible and authentic. However, three percent (3%) of the population reported that given the institutional repute in intricate research works, this research can feasibly be done locally.

Conclusion: Micro RNA's based study have taken place at this sub Saharan African University in the recent past especially in microorganisms at the University Molecular Laboratory. However, the possibility and feasibility of this study on breast cancer tissues is likely to be stalled in absence of a collaborating foreign research institution of repute where such research work is happening routinely.

Audience Take Away

- Breast cancer is a real women health threat in sub Saharan Africa.
- Disparities in cancer biology need a local research approach for best tailored control.
- Advances in research need to be tried in the various research site worldwide.
- International collaboration is the only likely solution for research feasibility in resource poor sub Saharan credible facilities.
- The utility of this study is empower global research interplay among researchers and funding agencies as a way of improving data on cancers with a worldwide generalized acceptability. A global approach to inform the scientific fraternity is only achievable when feasibility studies are under taken.

Collaborations with sub Saharan Scientists should always be sourced and emphasized at all fora of Cancer conferences starting with this very one if we are to fight cancer successfully and globally to address the disparities experienced.

Biography

Dr. Timothy studied Medicine at Mbarara University of Science and Technology, Uganda and graduated as MB ChB in 1998. He practiced Medicine up to 2002 then joined the Makerere University for residence in surgery for which is a Master's degree of Medicine in Surgery in 2005.He has been involved in a number of research activities at the Makerere University College of Health Sciences, (MakCHS). He has attained an MBA from Sikkim Manipal University of India in 2014, together with a Diploma in Emergency Medicine from the University of Rome, Sapienza in 2009. He is currently a PhD fellow at the same institution. He has supervised over 30 graduate research works and he is also a member of the School of Medicine Research and Ethics Committee of Makerere University, Uganda. He is also a practicing breast and endocrine specialist surgeon at the Makerere Teaching and National referral hospital, Mulago. He has obtained the position of a Senior Lecturer at the Department of Surgery of Makerere University. He has published more than 20 research articles in SCI (E) journals.)

Luis Filipe de Oliveira Figliolino*; Bottoni, A.
University of Mogi das Cruzes, Brazil

Carbohydrate loading: What science brings innovative to surgical oncology patients?

Hospital services have increasingly sought the satisfaction of their clients, through which we are able to measure the quality of health care, as well as other measures of behavioral outcomes of patients1. It is believed to have a direct relationship between the degree of satisfaction of the clients and the results against the proposals of treatment instituted. Studies comparing abbreviation routines of surgical fasting have long been described, and several of them have shown that abbreviating preoperative surgical fasting brings benefits for patients, such as accelerating postoperative recovery, attenuating the response inflammatory, improve the nitrogen and hydroelectrolyte balance, promote improvement in the immune response and healing, and also decrease the length of hospital stay. Regardless of nutritional status, reducing pre-operative fasting periods and increasing car- bohydrate loading can be beneficial in reducing surgically induced stress, attenuating insulin resistance, preserving muscle function and improving patient comfort. Combining nutritional measures in an ERAS programme can help reduce post-operative stay and complications. There is support for the use of immune-enhancing supplementation in certain population groups but more evidence is required. In the study I conducted during the preparation of my master's degree, I noticed that Carbohydrate loading is safe for patients undergoing elective surgeries. It is possible to prevent the onset of nausea and vomiting in the postoperative period when we take the patients from the state of prolonged fasting. Reduction of length of stay in one day. Save of hospital costs by approximately 13%. Surgeries with a higher degree of complexity (such as cholecystectomy for example), with shortened fasting, did not present complications. When we compared patients from this surgery who presented complications with those who did not present complications, the reduction in length of stay was greater and the reduction of hospital costs was reduced to 72%. However, nothing was more important than looking into my patient's eyes and feeling his gratitude for helping him go through something so traumatic for him in a better and safer way.

Audience Take Away

- Learn the importance of bringing your cancer patient safely.
- Show the importance of abolishing unnecessary prolonged fasting in hospital units.
- Bring to the public how important a health professional is in your life.
- Doing good does not matter to whom, and contributing to the recovery of the health of an audience that so much needs our knowledge.

Biography

Master's Degree in Science and Technology in Health from the University of Mogi das Cruzes. Postgraduate degree in Enteral and Parenteral Nutrition from GANEP - Human Nutrition. Currently working as a professor at Universidade Nove de Julho with teaching students in the internship field, a research area mainly on the following topics: sepsis, oxidative estrés, selenio; critical patient, pharmaconutrition, perioperative nutrition and short for fasting, nutritional care in the intensive care. He has published 6 research articles in SCI (E) journals.)

Melanie Di Benedetto[1,2,3*] , Guilhem Bousquet[1,2,3,6], Jean-Paul Feugeas[4], Christophe Leboeuf[1,2], Morad El Bouchtaoui[2], He Lu[2], Marc Espié[5], Anne Janin[1,2,7]

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Investigating the role of angiogenesis in chemoresistance-The example of API-5

Chemoresistance is a complex phenomenon involving intracellular as well as extracellular mechanisms that needs further studies. Among them, cell death inhibition or environmental cells (fibroblast or endothelial cells) interaction are still under studies. Anti-apoptotic protein-5 (API-5) is a survival protein interacting with the protein acinus, preventing its cleavage by caspase-3 and thus inhibiting apoptosis. 78 TNBC biopsies from patients with different responses to chemotherapy were analysed for API-5 expression before any treatment. Further studies on API-5 expression and inhibition were performed on patient-derived TNBC xenografts, one highly sensitive to chemotherapies (XBC-S) and the other resistant to most tested drugs (XBC-R). We studied the effect of targeting API-5 in chemoresistant triple negative breast cancers (TNBCs), to reverse chemoresistance.

Clinical analyses of the 78 TNBC biopsies revealed that API-5 was more markedly expressed in endothelial cells before any treatment among patients with chemoresistant TNBC, and this was associated with greater micro-vessel density. A transcriptomic analysis of xenografted tumors showed an involvement of anti-apoptotic genes in the XBC-R model, and API-5 expression was higher in XBC-R endothelial cells. API-5 expression was also correlated with hypoxic stress conditions both in vitro and in vivo. 28 days of anti-API-5 peptide efficiently inhibited the XBC-R xenograft via caspase-3 apoptosis. This inhibition was associated with major inhibition of angiogenesis associated with necrosis and apoptosis

Audience Take Away

- Angiogenesis in chemoresistance.
- Targeting endothelial cell precursors.
- PDX
- Apoptosis in chemoresistance.

The audience can take into account other ways to study chemoresistance and the possibility to identify new targets. Also the work is based on the use of patent tumors with different statues toward chemotherapy transferred into nude mice. Thus it could help audience in their design and acknowledgement in chemoresistance. For example, model to use other than those currently investigated (cell lines or chemoresistant induced one). The presentation offer a new approach in the understanding of chemoresistance through unknown pathways.

Biography

Dr Di Benedetto studied angiogenesiss in breast cancer at the Paris Sorbone university since 1999. She received her PhD degree in 2001 at the same institution After several year postdoctoral fellowship supervised by Dr clara Nahmias intitut cochin, France she obtained the position of an Associate Professor at the paris Sorbonne university. She joined the research group of Prof. Bousquet at INSERM, UMR_S942 MASCOT in 2010 and initiate to study translational breast cancer research including chemoresistance. She has published more than 40 research articles in SCI (E) journals.)

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DOT1L promotes Ovarian Cancer tumor growth by stimulating pro-tumorigenic metabolic pathways and blocking apoptosis

Ovarian cancer is the leading cause of gynecological malignancy-related deaths. Current therapies for ovarian cancer do not provide meaningful and sustainable clinical benefits, highlighting the need for new therapies. We show that the histone H3K79 methyltransferase disruptor of telomeric silencing 1-like (DOT1L) is overexpressed in ovarian cancer and that a higher level of DOT1L expression correlates with shorter progression-free and overall survival (OS). Pharmacological inhibition of DOT1L (EPZ-5676, EPZ004777, and SGC0946) or genetic inhibition of DOT1L attenuates the growth of ovarian cancer cells in cell culture and in a mouse xenograft model of ovarian cancer. Transcriptome-wide mRNA expression profiling shows that DOT1L inhibition results in the downregulation of genes involved in cellular biosynthesis pathways and the upregulation of proapoptotic genes. Consistent with the results of transcriptome analysis, the unbiased large-scale metabolomic analysis showed reduced levels of several metabolites of the amino acid and nucleotide biosynthesis pathways after DOT1L inhibition. DOT1L inhibition also resulted in the upregulation of the NKG2D ligand ULBP1 and subsequent increase in natural killer (NK) cell-mediated ovarian cancer eradication. Collectively, our results demonstrate that DOT1L promotes ovarian cancer tumor growth by regulating apoptotic and metabolic pathways as well as NK cell-mediated eradication of ovarian cancer and identifies DOT1L as a new pharmacological target for ovarian cancer therapy.

Audience Take Away

- Ovarian cancer remains a challenge for modern oncology practice because of its late stage detection, poor survival when the disease is metastasized, and lack of effective therapies. Identification and targeting of DOT1L can be employed as a new therapeutic intervention for ovarian cancer.
- New epigenetic regulator is involved in ovarian cancer growth and progression.
- Clinically relevant targetable mechanism for ovarian cancer therapy.

Biography

Dr. Gupta did her BS and MS in India. She then joined Prof. Knud Nierhaus group at Max Planck Institutefor Molecular Genetics, Berlin, Germany for her PhD and obtained her degree in the area of ribosome biology and protein translation. After that she worked at Yale University as postdoc where she extensivelyperformed studies to identify new regulator in cancer growth and progression. Many of her studies are published in journals like eLife, PNAS, Cell Reports, Oncogene etc. Currently she is an Assistant Professorin the UAB and Associate scientist at O'Neal Comprehensive Cancer Center at UAB. Her lab Our works onidentifying new molecules and pathways and studying their role in tumor initiation and progression. Her long-term goal is to not only identify new molecules and signaling pathways that regulate the disease but also develop more effective and durable cancer therapies.

Kaifu Chen[1,2,3,5*], Xinlei Gao[1,2,3,6], Yang Yi[4,6], Jie Lv[3,6], Yanqiang Li[1,2,3], Sahana Suresh Babu[3], Ivone Bruno[3], Lili Zhang[1], Qi Cao[4]

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RNA m6A landscape reveals strong regulatability of tumor suppressor expression

Cancer genes were known to display unique epigenetic features on chromatin of benign cells. Investigations into these features are making it increasingly clear that cancer genes differ from other genes regarding the mechanisms regulating their transcription. It is yet unknown whether cancer genes have a unique epitranscriptomic feature on RNAs and thus differ from other genes in post-transcriptional regulation of their RNA expression. Here we found RNAs of tumor suppressor genes tended to decay fast in multiple benign cell types when compared with other RNAs. Consistent with a negative effect of m6A modification on RNA stability, we observed preferential deposition of m6A on tumor suppressor RNAs. With frequent transcription, the fast RNA decay of tumor suppressors did not lead to low expression in benign cells. However, abundant m6A and fast decay of tumor suppressor RNAs both tended to be further enhanced in prostate cancer cells relative to benign prostate epithelial cells. This enhancement correlated with a down regulation of tumor suppressor expression. Further, knockdown of m6A methyltransferase METTL3 and reader protein YTHDF2 in prostate cancer cells posed stronger effect on tumor suppressor RNAs than on other RNAs. These results indicated a strong expression maneuverability of tumor suppressors mediated by abundant m6A modification on RNAs.

Audience Take Away

- Transcripts of tumor suppressor genes tend to have a low stability and thus decay fast in benign cells.
- The low stability of tumor suppressor RNAs is conserved across benign cell types.
- The low stability of tumor suppressor RNAs is mediated by preferential deposition of m6A modification on these transcripts in the cells.
- The m6A-mediated low stability of tumor suppressor RNAs is further enhanced in cancer cells relative to normal cells.
- Compared with other RNAs, tumor suppressor RNAs are more sensitive to manipulation of RNA stability regulators, e.g., the m6A "writer" protein METTL3 and "reader" protein YTHDF2.

Biography

Kaifu received Ph.D. training in Genomics at the Beijing Institute of Genomics, Chinese Academy of Sciences. He performed postdoctoral research at the Baylor College of medicine. He then joined the Houston Methodist Hospital and Weill Cornell Medical College as an Assistant Professor, and later became the Director of their Center for Bioinformatics and Computational Biology. He finally moved to Harvard Medical School at Boston Children's Hospital as an Associate Professor and is now directing their Computational Biology Program. Kaifu's major research interest is in computational modeling of how cell identity is established, maintained, and dysregulated.

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Tamoxifen : Contraceptive pill to anti-cancer drug

Tamoxifen, an anticancer drug which acts by competitive binding to estrogen receptor by blocking G1 phase of cell cycle to slow cell proliferation by reduction of transcription of estrogen regulated genes.

Tamoxifen which was synthesized in 1962 and first discovered as compound ICI 46,474 used as an anti-estrogen to prevent ovulation in women.After completing some Clinical trials, it has been showed that it could prevent its occurance in women at high risk of developing breast cancer. Tamoxifen is a perfect example of pharmaceutical evaluation of drug which discovered as a contraceptive pill but used widely to prevent breast cancer by its pharmacological action. After discovering as a contraceptive, It was very difficult to prove that tamoxifen has a prominent action in palliative treatment for patients with breast cancer. As per initial results in 1983, The Lancet reported that Tamoxifen helped 10-20% more women over age 50 to survive for cancer in 2-3 years after surgery and radiation. Additional results published in 1985 proved that 6 years treatment of tamoxifen can reduce death 34% for patients of all ages with the disease.

However in the treatment of breat cancer, 5 years treatment of tamoxifen shows more effectiveness than lesser time treatment.

Audience Take Away

- Tamoxifen: It's anti-cancer effects.
- Tamoxifen: Evaluation to Anticancer drug.

Biography

Mr. Soumik Laha, Assistant Professor of Bengal College of Pharmaceutical Sciences & Research, Durgapur which is located in West Bengal, India.He has completed his M.Pharm in Pharmaceutics from JNTU Hyderabad.

Shivi Mudgal[1]*, Dr Neel Kamal Arora[2]

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Lymphatic drainage of Breast: Clinicoanatomical perspective in Carcinoma Breast

One of the most frequent types of neoplastic diseases in females worldwide is breast cancer resulting in significant morbidity and mortality. The commonest route of spread of breast tumor cells is vialymphatics and blood.

We as anatomists would like to impress upon the lymphatic anatomy of breast. One of the cardinal factorsdetermining the clinical presentation, diagnosis, staging, management and prognosis of carcinoma breast isthe knowledge of involved lymph nodes – the area they drain, their anatomical location, adjacent relationsand connections. The breast cancer cells metastasize to regional lymph nodes and thence travel to distant sites giving rise to secondaries. The speaker would like to take this opportunity to describe the lymphaticsof mammary gland. The lymphatic drainage of parenchyma of the breast and the overlying skin will be discussed followed by the anatomical basis of clinical presentation of breast cancer. The anatomical and surgical classification of lymph nodes draining the breast tissue will be detailed along with their locations,connections and anatomical relations.

The knowledge helps in clinical examination of axillary, supraclavicular and parasternal lymph nodes. It helps the oncologist to plan diagnostic and therapeutic interventions, predict the potential sites of secondaries by identifying the major routes of lymphatic metastasis, and explains the clinical experience inmapping the breast lymphatics, sentinel lymph node biopsy and axillary lymph node dissection. Knowing the anatomical location of sentinel node helps in focussed surgical dissection and radiotherpy thereby reducing post operative morbidity and lymphedema.

Biography

Dr. Shivi Mudgal studied Human Anatomy at Maharishi Markandeshwar University, Haryana (India) and graduated as MD in 2013, securing first position in University Examination. She joined VMMC and Safdarjung Hospital, New Delhi as Senior Resident in Anatomy for 3 years. She is presently working as Assistant Professor (Anatomy) in SRMS Institute of Medical Sciences, Bareilly. She has several publications in PubMed indexed international journals. She actively participates in national and regional conferences, workshops and webinars and is life member of Anatomical Society of India and Society of Clinical Anatomists. Her fields of interest are Gross Anatomy, Radiological Anatomy, Developmental Anatomy and Cadaveric Dissection.

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Awareness of Oral Cancer in a community of Sunsari, Nepal: Oral Cancer awareness saves lives

Introduction: One of the most prevalent cancers in the world, oral cancer is also one of the leading causes of mortality in some areas, such as South-Central Asia, and is a significant public health issue. Oral cancer is the third most prevalent cancer in women and the most common cancer in men in Nepal, according to the National Health Policy. Several studies have shown that the public is not well aware of the risk factors associated with oral cancer. The objective of this study was to assess the awareness of oral cancer among people of Sunsari.

Methodology: A cross-sectional descriptive study was conducted among people in the age group 18-65 years in 2019. Data were collected from 128 respondents through face-to-face interviews using Semi – a structured interview schedule with a non-probability purposive sampling method. Data entry and analysis were done in SPSS version 17. Descriptive statistics i.e., frequency, percentage, the mean, and standard deviation were used to assess the level of knowledge and inferential statistics i.e., chi-square was used to find the association between awareness of oral cancer and selected demographic variables.

Results: Findings of the study showed that more than half (59.4%) had heard of oral cancer regarding the source of information, 71.1% had stated that media. The majority (89%) said oral cancer was not contagious and more than half (52.3%) of respondents were known about treatment modalities for Oral cancer whereas two third, 78.9% of respondents stated out movement is best for physical rehabilitation. Likewise, more than half (65.6%) had a moderate level of awareness and 28.1% of the respondents had an inadequate level of awareness regarding oral cancer. The result showed there was no association between the level of awareness of oral cancer and selected demographic variables.

Conclusion: The study concluded that awareness regarding oral cancer is moderate among the community people of Sunsari, Nepal. It reflects that efforts should be made to increase awareness regarding oral cancer. Health education regarding oral cancer and its risk factors with periodic reinforcement will play an important role in creating awareness. Furthermore, systematic awareness programs about the role of habits in the development of oral cancer, its complication, and the benefits of detecting it at an early stage need to be implemented by policymakers, institutions, and hospitals for a better outcome.

Keywords: Awareness, oral cancer, community people

Audience Take Away

- Our findings have several implications for institutions, hospitals, and policymakers. The audience will be able to learn about the level of awareness of oral cancer in a community and they will use these findings in developing countries facing similar problems. The findings will help them to design and initiate an intensive public education program for the recognition of early warning signs of oral cancer and early detection by mouth self-examination. Furthermore, it helps future researchers to gain methodological insight and conduct a comparative study among urban areas and rural areas.

Biography

Ms. Punam Kumari Mandal is a faculty of Tribhuvan University Institute of Medicine. She graduated MPH from the Institute of Medicine, Nepal in 2013. She involved in teaching research, biostatistics, and supervised research work for more than 8 years. She is a member of the research committee at this institute. She has been involved in quantitative and qualitative as well as collaborative research. She has been awarded the gold medal by the President of Nepal. She received Nepal Bidhya Bhushan and Nepal Chatra Bidhya Padak

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Evolution of androgenic deprivation in treatment of Prostate Cancer at Kinshasa, DRC.

Context and objective: Prostate cancer (PCa) is hormone-dependent cancer. In our area, most patients often arrive at the locally advanced stage or the metastatic stage. This justifies the choice of androgen deprivation as the mode of treatment. The objective of this study was to describe the socio-demographic characteristics of patients with PCa. Identifying the period during which the disease remains susceptible to androgen deprivation. Assessing the patient's prognosis in terms of survival.

Methods: This is a retrospective observational study of the course of patients managed for PCa. It involved 51 cases and was conducted at the Pointe à Pitre clinic (CPAP) in Matete Township during a period of 4 years (from March 2014 to June 2018).

Results: The mean age of patients was 69.4 ± 9.7 years (40-92 years); 39.2% of patients with PCa were aged between 70-79 years; 45.1% had consulted for dysuria and 25.5% were hypertensive. All had performed the prostate biopsy, 47.1% were diagnosed at the metastatic stage, with PSA ≥100ng/ml, Gleason scores 8-10, and clinical-stage TNM3-4. About 51% were subjected to androcure, 23.5% had been surgically cased and 3.9% had undergone radical prostatectomy. 41.1% had resisted castration within a median of 1.4, [1-3] years of response to treatment. The median survival was 30 months, with a mean survival of 26.6 months.

Conclusion: Prostate cancer involved most of the patients in the age bracket of 70 to 79 years. The diagnosis was performed lately with a high resistance rate of castration and median survival of 30 months.

Keywords : Deprivation, Prostate cancer (PCa), Pointe à Pitre Clinic (CPAP).

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Pharmacovigilance in Multiple Myeloma Care in sub-Saharan Africa

The quality and disability adjusted life-years are the two predictive markers of average life expectancy of any patient. In Multiple myeloma (MM), the duos are the functions of the stage of presentation of primary disease and the impact of the therapeutic (anti-myeloma) intervention. The ''impact of therapeutic intervention'' in this context connotes the safety and the adverse effects of anti-myeloma target drugs on the body. The ability to detect or predict the adverse effects of anti-myeloma target therapy and protect the patient or prevent the occurrence will contribute to improved life expectancy of target MM patient. Pharmacovigilance on anti-myeloma therapy is the science that deals with risk assessment and management of prospective anti-myeloma target drugs. This framework ensures safety and surveillance of anti-myeloma drugs administered to prospective MM patients. It is a very important arm of comprehensive care of people living with MM. This study highlights the safety measures undertaken by the healthcare professionals and MM patients who are on immunomodulatory anti-myeloma drugs in order to minimize adverse drug reactions in the underserved areas of sub-Saharan Africa.

This study was an integrated review of the works of literature on pharmacovigilance of a MM patients on anti-myeloma chemotherapeutic agents and the risk management programme for immunomodulatory anti-myeloma target drugs (thalidomide and lenalidomide) using Pub Med, Medline, CINAHL, Google Scholar, AJOL and pharmacovigilance practical manuals from African countries as databases. The paper was analysed and grouped according to the following categories namely an introduction to pharmacovigilance in the care of MM patients, some of the adverse drug reactions encountered with the use of anti-myeloma drugs, pharmacovigilance risk management programme and risk management protocols for immunomodulatory drugs.

This study showed that an effective pharmacovigilance programme requires specially trained healthcare professionals made up of physician (prescriber), pathologist, pharmacist (dispenser), oncology director (haemato-oncologist), nurse, social worker and palliative care specialist. The consent/registration adverse drug reaction forms are the essential safety working documents that are needed to be completed by the patient and healthcare professionals. Both healthcare provider and the patient are expected to adhere strictly to the safety measures enshrined in the healthcare professional and the patient's pharmacovigilance brochures respectively. The patient's anti-myeloma safety measures are stratified into female of child bearing age, women above the child-bearing age and men. The essential safety requirements include signing treatment initiation form and avoidance of blood donation during treatment. Other requirements stipulate that the female must be birth control compliant, pregnancy negative and must not breastfeed during treatment with thalidomide and lenalidomide, while the man must use condom during sexual activities while on treatment.

In conclusion, Pharmacovigilance in the care of people living with MM is a strategic leadership approach of maximizing the quality adjusted life-years while minimizing the disability adjusted life-years of target population at the same time using safety measures. The hallmark is increase in average life expectancy of MM patients. In order to establish an effective pharmacovigilance program, it requires human capacity development, sustainable drug availability, collaboration with support groups, and policy formulation.

Audience Take Away

- They will appreciate the role of pharmacovigilance as a health protective mechanism in cancer management vis-a-viz multiple myeloma.
- They will appreciate pharmacovigilance as adverse drug reaction preventive approach in cancer management vis-a-viz multiple myeloma.
- They will learn the importance of setting-up a risk management protocol (RMP) in cancer treatment.

Biography

Dr. Ogbonna Collins NWABUKO is an Assistant Professor (MD, FMCPath) at the Department of Haematology, University of Calabar, Cross-Rivers State, Nigeria. He holds a Master of Science in Public Health from University of South Wales, and specialty training in Palliative Medicine. He is a member of Nigerian Society of Haematology and Blood Transfusion, American Society of Haematology, American Public Health Association and African Palliative Care Association. He is also a fellow member Eudoxia Research University, USA (FMERU). He is the principal investigator of ''the burden of Multiple Myeloma in Nigeria. He has over 75 research articles in SCI (E) journals, authored two books, two chapters and reviewed sixty-one manuscripts including Sage and Lancet Haematology.

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POSTERS

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Andrea Martisova[1,2]*, Lucia Sommerova[1], Iveta Selingerova[1], Adam Krejci[1], Tamara Kolarova[1], Filip Zavadil Kokas[1] and Roman Hrstka[1]

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Identification of gene-specific signatures associated with AGR2 expression and TGF-β exposure in Lung Cancer cellular model

Epithelial to mesenchymal transition (EMT) is one of the key processes in tumour progression and metastasis development. TGF-β signaling plays an important role in this process, and through a cascade of signaling events, it enhances the transcription of EMT drivers. Recently, AGR2 protein emerged as a crucial component of the cellular machinery responsible for maintaining epithelial phenotype, hence interfering with the TGF-β pathway. We performed transcriptomic profiling of A549 lung cancer cell line with CRISPR-Cas9 mediated *AGR2* knockout together with and without TGF-β treatment. The obtained results were subjected to PANTHER and DAVID analysis. Significant changes were identified in transcripts associated predominantly with focal adhesion and arachidonic acid metabolism. These changes were then validated on the selected panel of transcripts using RT-qPCR. Moreover, immunofluorescent staining showed induced formation of stress fibres containing vinculin foci in cells without AGR2 and in response to TGF-β treatment. Transcripts associated with arachidonic acid metabolism showed decreased levels after both *AGR2* gene knockout and exposure to TGF-β and likewise were validated by RT-qPCR. Since prostaglandin E_2 (PGE_2) is a product of arachidonic metabolism, its lowered concentration in media after *AGR2* gene knockdown and TGF-β treatment was confirmed by ELISA. In contrast, the addition of exogenous PGE_2 significantly elevated AGR2 protein level in lung A549 and colorectal HT29 cancer cell lines. Together, our results imply that AGR2 downregulation and TGF-β have an essential role in focal adhesion formation; moreover, we have newly identified AGR2 as an important component of the arachidonic acid metabolic pathway. This work was supported by MH CZ - DRO (MMCI, 00209805) and European Structural and Investment Funds - Project “Mobility in MMCI” (No. CZ.02.2.69/0.0/0.0/18_053/0017836).

Audience take away

- This work broadens the knowledge about AGR2 and TGF-β impact on gene expression in lung cancer cell line.
- Deeper knowledge concerning the epithelial-mesenchymal transition expression patterns with respect to AGR2.
- AGR2 is a component of focal adhesion pathway.
- AGR2 is a component of Arachidonic acid metabolism pathway.

Biography

Andrea Martisova is a doctoral candidate at Masaryk University (Brno, Czech Republic), where she also gained her Master´s degree in the field of Genomics and Proteomics in 2020. She has been conducting her research in the Research Centre for Applied Molecular Oncology at Masaryk Memorial Cancer Institute (Brno, Czech Republic) since autumn 2017. The majority of her work is focused on the role and regulation of the Anterior Gradient 2 (AGR2) protein in cancer cells. As a part of her doctoral program she is currently undertaking an internship in the “Oncogenesis, stress, Signaling” (COSS) Inserm U1242 unit in Rennes.

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JAK 2 negative Polycythemia vera in tertiary care center in West UP population in India: A rare presentation of 3 cases.

Polycythaemia vera (PV) belongs to the group of myeloproliferative neoplasms with excessive increase in hemoglobin levels. They are clinically characterized by nonspecific symptoms such as fatigability, pruritus, early satiety due to splenomegaly, increased risk of infections, and thrombotic events. JAK2 V617F mutation present in 80-90% of MPNs and JAK2 exon12 mutations are seen in 4%-5% cases of MPNs like PV. Activation of JAK2 by either point mutation or fusion protein causes activation of the JAK-STAT pathway. While mutations in JAK2 are reported in numerous MPN phenotypes, exon 12 mutations specifically result in erythrocytosis due to increased EPO signaling.

Case series

Case 1-A 21-year male presented to the hematology OPD with complains of parasthesias and off and on headache for the past 6 months. He was on multiple symptomatic medications but to no relief. On routine investigations it was seen that he had a persistent hemoglobin level fluctuating between 17.5gm/dl and 18.5 gm/dl. He has iron deficiency for which he is on single oral iron tablet. No definite organomegaly was seen. In hematology OPD he was investigated for polycythemia vera keeping in mind the WHO criterias. His Serum EPO levels are within normal range,JAK2 V617F/exon 12mutation analysis is negative. A bone marrow aspiration and biopsy study done shows Hypoplastic marrow for age with erythroid predominance. In view of his clinical presentation he is being treated with ecosprin, Supplemental iron orally and phlebotomy every 3 months and patient is clinically relieved of his symptoms. His last hemoglobin value is stable at 16.5gm/dl.

Case 2-A 32 -year male presented to hematology OPD with mild headache and weakness for 6 months. A routine CBC investigation showed a high hemoglobin level of 18.4 gm/dl. Mildly raised RBC count was also seen. Rest all parameters were within normal limits. Similar to the previous case no relevant examination findings were there. A subnormal serum EPO levels of 3.7 mIU/Ml (4.3-29.0 mIU/ML) was seen. JAK 2V617F/exon 12 mutation analysis was negative. The patient has been started on ecosprin and phlebotomy every 3 months and is symptomatically relieved.

Case 3- A 29-year male presented to hematology OPD with paresthesias and weakness for 3 months. A routine CBC investigation showed a high hemoglobin level of 19.7 gm/dl. Mildly raised RBC count was also seen. Rest all parameters were within normal limits. Similar to the previous case no relevant examination findings were there. A low serum EPO levels of <1 mIU/Ml (4.3-29.0 mIU/ML) was seen. JAK 2V617F/exon12 mutation analysis was negative. The patient has been started on ecosprin and phlebotomy every 3 months and is symptomatically relieved.

Conclusion-All three cases that presented to the department had been undiagnosed for 2-3 months before they reached our setup. The unique feature of all these cases was negativity for both JAK2V617F as well as JAK2 exon 12 mutation despite its clinical behavior being that of PV .These cases are being actively managed as low risk polycythemia vera and since the start of the treatment comprising of phlebotomy once every 3 months their hemoglobin and hematocrit concentrations are maintained. No cytoreductive therapy is being given .Such unique presentations need to be reported and discussed extensively.

Audience Take Away

- Learning point in the above case series
  1. As far as the spectrum of MPNs go we strictly follow the WHO criteria for diagnosis but the field of oncology and hemato-oncology is dynamic and always evolving hence we are slowly seeing a rise in cases which might not have a defined molecular and cytogenetic profile but are clinically benefitting from treatments.

  2. In our case the patients were treated with therapy modality for PV and benefitted.

  3. Hence these cases alert the pathologist and the clinician to widen their thoughts while looking at cases of MPNs specifically PVs.
- I am extremely interested in research specially in the field of hematology and work closely with clinical hematologists hence this will benefit research and therapy equally to highlight such cases so that clinicians know that there are a percentage of cases which might not look what they seem.

Biography

Neema Tiwari did her MBBS and MD in Pathology in Pathology from Eras Lucknow Medical college and Hospital, She is has worked as Senior Resident, Pathology in department of clinical hematology and hemato-oncology, King George Medical University, India and Post Graduate Institute of Child Health Noida, UP and has 4 years post PG experience. She is currently working as Assistant Professor in Subharti Medical College Meerut. She has done numerous intarmural and extramural (ICMR,DST)research projects and has many national and international publications in indexed and peer reviewed journals(>40) to her credit. She is a reviewer for 3 journals to of which are pubmed indexed. She has presented papers in IAP,ICC and CAP conferences. She has recently presented a poster on MDS in the ISHBT-EHS TUTORIAL-2018 held in India.

She has also co-authored 2 books titled

A) Analysis of various patterns of leukemia in Indian population-Neema Tiwari, Sunita Tiwari (Lambert publications, now available in Barns and Noble website, Amazon,flipkart)-2018

B) Manual of Hematology, Ahuja publishers-2021

C) 3 Book chapters in 2 books under publication-2021

She has her YouTube channel for teaching Pathology to postgraduate residents in her name with >1500 followers

She in International ambassador for College of American Pathologists in India for current 2 years

She is an International Observer (Pathology under Dr Travis and Dr Antenescou) alumni in Memorial Sloan Kettering Cancer Centre, New York, USA

Member European hematology association, Junior Member CAP, Member ISHBT,Member ESP

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The direct and indirect effects of affluence on survival in Colon Cancer: A population-based study

Background: Colorectal cancer is the fourth most common cancer in the UK. Socio-economic disparities are apparent in colorectal cancer survival in the UK and elsewhere. However, it is not known whether socio-economic status has a direct influence on survival or whether it operates via other factors, such as stage. We investigated the direct and indirect effects of deprivation on colon cancer survival.

Methods: Cases of colon cancer [ICD10 C18] diagnosed 2001-2010 were identified from the Northern and Yorkshire Cancer Registry. Deprivation was based on the IMD income domain of the area of residence at diagnosis. Using a discrete-time survival model within a structural equation modeling-type approach, the direct and indirect effects of deprivation, and other explanatory variables, on the hazard of all-cause mortality were analyzed.

Results: 31,779 cases of colon cancer were included. Those resident in more affluence areas had significantly earlier stage of disease, received optimal (rather than sub-optimal) treatment and started treatment promptly (≤31 days from diagnosis). Affluence significantly and directly reduced the hazard of mortality. Optimal treatment, female sex, and more recent year of diagnosis also directly reduced the hazard while increasing age, untimely treatment and later stage directly increased it. The hazard of mortality was reduced indirectly by affluence, mediated by increased optimal treatment, and reduced untimely treatment and late stage.

Conclusion: These findings demonstrate that deprivation has a direct effect on survival in patients with colon cancer. Moreover, patients resident in deprived areas are further disadvantaged by sub-optimal and untimely treatment.
This study was funded by Cancer Research UK and the NIHR School of Primary Care Research.

Audience Take Away

- This poster will increase the understanding of how cancer survival is affected by social deprivation. The use of a discrete-time survival model within a structural equation modelling-type approach could be used by other researchers on other datasets to enhance the understanding of direct and indirect effects.

Biography

Dr Rosie Dew gained her PhD at Newcastle University, UK in 2015 and has worked as a postdoctoral research associate at both the University of Sunderland and Newcastle University on a range of health science projects. She currently works as a Senior Lecturer in Physiology at the School of Medicine, University of Sunderland.

Sara Mikhael[1], Kurdi A[2], Zgheib N K[3], Tahtouh R[1], Nasr R[4]*, Hilal G[1]***

[1]Laboratory of Cancer and Metabolism, Faculty of Medicine, Saint-Joseph University, Lebanon.
[2]Department of Biochemistry and Molecular Genetics, American University of Beirut, Lebanon.
[3]Department of Pharmacology and Toxicology, Faculty of medicine, American University of Beirut, Lebanon.
[4]Department of Anatomy, Cell Biology and Physiological Sciences, Faculty of Medicine, American University of Beirut (AUB), Lebanon
*Co-corresponding authors

Outlining the synergistic effects of combining Metformin and Simvastatin in Ovarian Cancer cells

Background: Improving response rates in Ovarian cancer is an urgent clinical issue. Drug repurposing offers a safe and a relatively fast alternative to the conventional methods for oncological drug discovery. Both, metformin, a biguanide approved to treat diabetes, and simvastatin, an HMG-CoA reductase inhibitor approved for hypercholesterolemia, are currently being researched in oncology. However, in vitro and in vivo studies have not yet been implemented on this combination (or any kind of statin) in ovarian cancer. The aim of the study is to elucidate the molecular mechanisms of the potential synergy ofcombining the metabolic altering drugs, metformin and simvastatin, in ovarian cancer cells.

Methods: OVCAR-3 cell line was used in all methods. First, we compared the concentrations of metformin MET and simvastatin SIM using a WST-1 assay. Conditions were as follows: untreated samples as control;MET (0 – 100mM); SIM (0-100μM). Second, we conducted a synergy analysis for both drugs using CompuSyn software. Third, we performed a transcriptome analysis using GeneChip™ WT PLUS Reagent Kit. The microarray data was analyzed using customized R/ Bioconductor pipeline.

Results: Cytotoxicity analysis aided in the choice of downstream treatment concentrations as such: MET 10 mM and sim 5μM. The combination of MET and SIM synergistically inhibited the proliferation of OVCAR-3 cells. Transcriptomic data comprised 1722 differentially expressed genes (DEGs). 507 DEGs were exclusively found in the combination arm with the top 5 most significant downregulated DEGs being: ABI1,AP1B1, TROAP, SPEF1, CLDN14 and upregulated being: CEP19, TMPO, TGOLN2, CTTNBP2, AWAT2.

Conclusion and outlook: Collectively, our data suggest the presence of synergy between both drugs in OVCAR-3. Further data analysis to identify enriched pathways and validation of the top DEGs by real timePCR are still in progress.

Audience Take Away

- Metformin and Simvastatin are anti-cancer agents.
- Metformin and Simvastatin are synergistic in Ovarian Cancer.
- Metformin and Simvastatin target metabolic and non-metabolic signaling pathways through various targets revealed through transcriptomic analysis.
- The data presented may be a basis for trials combining Metformin and Simvastatin with standard chemotherapeutic medications to study further synergy.

Biography

Sara Mikhael obtained her bachelor's degree in pharmacy from the Lebanese American University. With high distinction, she obtained her Master's degree in Cancer and Therapeutics at the Barts Cancer Institutein London with a thesis entitled "Impact of tumor heterogeneity on the response to kinase targeted therapies". She is currently completing her doctoral studies in medical and biological sciences at L'Université Saint-Joseph de Beyrouth (USJ) in collaboration with the American University of Beirut (AUB).Her project focuses on the use of metabolism-altering drugs, simvastatin and metformin, in ovarian cancer, drugs that are not typically used in cancer therapy.

Parul Sharma*, Siddharth Sharma
Department of Biotechnology, Thapar Institute of engineering and technology, India

Polymorphism in Thymidylate Synthase gene predicts survival and toxicity in North Indian Lung Cancer patients undergoing platinum-based doublet chemotherapy

Lung cancer is one of the most widely reported tumors having high morbidity and mortality rates with an estimated 1.8 million fatalities. Thymidylate synthase (TS) is an important target for chemotherapy treatments and platinum-based therapies since it is the cell's only de novo source of thymidylate production. TS polymorphisms in the TS enhancer region (TSER) 2R/3R and TS 1494del6 in the 5' and 3'-untranslated regions (UTRs) are investigated in this study. A total of 700 lung cancer patients with platinum-based chemotherapy were recruited in the study. Polymorphisms of TSER (2R/3R) and TS 1494del6 in North Indian lung cancer patients were assessed and statistical analysis were carried out. Our data observed that patients with wild genotype (2R/2R) for TSER polymorphism showed higher trend of median survival time compared to patients bearing the mutant type genotype (3R/3R) [MST=9.77 vs. 7.57 months; p=0.04]. Patients with mutant genotype (-6/-6) for 14946del6 polymorphism showed higher survival time compared to patients bearing the wild type genotype (+6/+6) [MST=7.23 vs. 9 months]. Further, we observed that the patients with heterozygous genotype (2R3R) for TSER polymorphism had a 2.30-fold increased risk of developing leukopenia (AOR=2.30, 95% CI=0.96-5.52; p=**0.05**) when compared to the subjects with wild type genotype (2R2R). A substantial risk of 5.14-fold constipation was found in heterozygous genotype (2R3R) when intermediate grade 2 toxicity was compared with low toxicity (grade 1) (p=**0.007**).An increased risk of nausea/vomiting was observed in patients with mutant genotype (-6/-6bp) for 1494 ins/del6 polymorphism compared to patients with wild-type genotype (+6/+6bp) (AOR= 2.77; 95%CI=1.10-6.96, p=**0.03**). Our data suggest that TSER and 1494del6 polymorphism act as a predictive marker in lung cancer patients treated with platinum chemotherapy. Also, TS polymorphisms might impact the development of platinum-related toxicities such hematological and gastrointestinal toxicity. These findings might facilitate therapeutic decisions for individualized therapy in lung cancer patient.

Keywords: Lung cancer, Thymidylate synthase, survival, platinum-based chemotherapy, polymorphism, Toxicity

Biography

Miss Parul Sharma is a research scholar under the supervision of Dr. Siddharth Sharma in biotechnology department in TIET, Patiala, India. Currently, I have published 9 research articles in SCI (E) journals.

Dr. Ahmad Ijaz Masood, Dr. Rabeeta Sheikh, Dr. Rana Atique Anwer, Dan Kenner[1]*

[1]ImunoBran, Luxembourg and France

"BIOBRAN MGN-3"; Effect of reducing side effects of chemotherapy in Breast Cancer patients.

The aim of study was to assess the effect of Biobran in reducing of chemotherapy induced side effects in terms of tiredness, anorexia, vomiting and hair loss and quality of life in terms of weight loss. Setting: Radiotherapy Department, Nishtar Hospital Multan. Material and Methods: Fifty patients of breast cancer were enrolled randomly in two groups. Group-A patients were given 3 gram dose of Biobran MGN-3 per day one week before and one week after chemotherapy. Group-B patient were given chemotherapy alone. Total six cycles of chemotherapy were given. No multivitamin or food supplements were given during this study. Chemotherapy induced side effects (tiredness, anorexia, and vomiting, hair loss) were assessed by questionnaire to the patients before start of each cycle. Weight was checked before each cycle to assess weight gain or loss. White blood cells were checked by complete blood count just before and one week after chemotherapy. Results: Between six months, 50 patients were enrolled in Radiotherapy Department, Nishtar Hospital Multan. There was a significant reduction in tiredness and anorexia in group-A patients. 20 (80%) patients of group-A felt increase in their diet and no tiredness without any appetizer or multivitamin. But group-B patients demanded for appetizer due to severe anorexia after chemotherapy except 3 (12%) patients who didn't use any appetizer or food supplement. In group-A, 15 (60%) patients didn't need any anti-emetic as compared to group-B all patient (100%) experienced severe nausea during and after chemotherapy. Group-A patients experienced less hair fall 7 (28%) patients as compared to other group which is 25 (100%) patients. Conclusions: The study showed that, by helping to optimize the immune system, Biobran MGN-3 can not only help maximize treatment success, but also minimize treatment side effects and improve quality of life during treatment and in recovery.

Biography

DAN KENNER, Ph.D., L.Ac graduated in 1979 from the Meiji College of Oriental Medicine in Japan, passed the Japanese National Licensing Examination and then trained in Internships at Osaka Medical University Pain Clinic and Kinki University Medical Teaching Hospital. He is licensed to practice Oriental Medicine both in the U.S. and in Japan. He also has a Ph.D. in Naturopathic Medical Science from the First National University of Naturopathic Medical Sciences. Dr. Kenner is on the Board of Directors of the Acupuncture and Integrative Medicine College in Berkeley, California and the National Health Federation. He is author of The Whole-Body Workbook for Cancer and other titles. Since 1983, he has endeavoured to integrate the Naturopathic Medical Traditions of North America and Europe with the Traditional Medicine of East Asia.

KEYNOTE FORUM

DAY 02

5TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

16-17 SEPT

Shinya Tajima[1]*, Keiko Kishimoto[2], Masayuki Takagi[3]

[1]Department of Diagnostic Pathology, National Hospital Organization Shizuoka Medical Center, Japan,
[2]Division of Radiology, Nihon-Bashi Arayla Clinic, Japan
[3]Department of Diagnostic Pathology, National Hospital Organization Shizuoka Medical Center, Japan

Muc-mucins of special feature in "nuclear inverse-polarity papillary lesion lacking myoepithelial cells"

Breast papillary lesions exhibit broad range. Tajima et al. reported in discrimination between benign intraductal papilloma (IDP) and malignant endocrine ductal carcinoma in situ (E-DCIS), new marker of CD56 is useful for discriminating between benign and malignant. Hence, detecting benign IDP correctly and exclude malignant lesion is important thing in daily pathological diagnoses. Here, in relation to IDP, we would like to present new concept of two papillary lesions at a glance IDP. In the past, lacking myoepithelial cells is thought to be invasion and means malignancy. We will present distinctive histological subtype which pathologists over-diagnose malignant however benign truly. Now we demonstrate two cases of 68- (Case1) and 44-year-old (Case2) female. They have abnormality in the breast. And they came to the hospital for further examination and treatment. Radiologically, malignancy could not be completely excluded. Then, breast excision was performed. Histologically, both cases revealed papillary neoplastic lesions lined by fibrovascular core and nuclear inverse-polarity without atypia. Loss of myoepithelial cells was observed by HE, p63, and calponin. Previous report indicate CK5/6, ER, p63 and MUC3 are important for distinguishing between papillary lesions according to the differential index (based on Allred score) of ([ER total score] + [MUC3 total score]) / ([CK5/6 total score] + [p63 total score] + 1). Based on this analysis, our 2 cases had benign lesions. Additionally, the Ki-67 index was <1% in both cases, and no evidence of disease was observed minimum 62 months of follow-up for both cases, despite lack of additional treatment. Here, we newly experimented MUC immunostainings in these cases because MUC status is important in breast diseases. We did immunostaining of MUC1,2,4,5AC,5B and 6. The results are MUC2,4,5AC and 6 are negative. MUC1 revealed apical strong staining and also MUC5B was completely negative. MUC1 of apical staining is thought to be benign. MUC5B is thought that the staining positivity means early cancer lesion. Hence our staining status also turn out to be benign without myoepithelial cells. In conclusion, MUC immunostaining status also proved "Nuclear inverse-polarity papillary lesion lacking myoepithelial cells" are benign lesions. Our lesion is histologically distinctive and the term of name is long, also exhibit special biological behavior, someone think "Tajima tumor" might be appropriate.

In this congress, I emphasize lacking of myoepithelial cells does not necessarily indicate malignancy. And I think it will be increase new histological subtype of benign however without myoepithelial cells. This notion is new concept and I would like to discuss with researchers in this congress.

Audience Take Away

- Through our new histological subtype, we can reduce unnecessary operation.
- If our tumor is commonly diagnosed, it would be better for breast tumor patients.
- Our knowledge will discover commonsense and contribute to daily pathological diagnoses.
- In the breast, lactifellous duct is maintained by two cells of cell-cell interaction. One is luminal cell and the other is myoepithelial cell. Usually myoepithelial cells control luminal cells for their invasion. However the two cells of cell-cell interaction disordered, invasive carcinoma arise from the lactifellous duct. Hence, lacking myoepithelial cells are considered invasion and malignancy. However our cases are indicated benign however lacking myoepithelial cells. To learn our new distinctive histological subtype, we can reduce unnecessary operation and increase patients' Quality of Life.

Biography

I am Shinya Tajima MD, PhD from Japan. I graduated Keio University School of Medicine. After graduated the university, working in Department of Pathology at the same institution. I learned general pathology. Then I would like to be a specialist of breast pathology. I affiliated St. Marianna University which is the most breast operation number in Japanese university. I received PhD in Radiologic-Pathology from the same Graduate School of Medicine, Kanagawa, Japan. I worked at the Department of Pathology and Radiology of this latter institution. Now I am working at Department of Diagnostic Pathology of Shizuoka Medical Center.

George Zachos

Department of Biology, University of Crete, Greece

The abscission checkpoint: A protector of chromosomal stability

The abscission checkpoint contributes to the fidelity of chromosome segregation by delaying completion of cytokinesis (abscission) when there is chromatin lagging in the intercellular canal between dividing cells. In mammalian cells, the abscission checkpoint requires proper localization and optimal kinase activity of the Chromosomal Passenger Complex (CPC)-catalytic subunit Aurora B at the midbody and culminates in the inhibition of Endosomal Sorting Complex Required for Transport-III (ESCRT-III) components at the abscission site to delay the final cut. Furthermore, cells with an active checkpoint stabilize the narrow cytoplasmic canal that connects the two daughter cells until the chromatin bridges are resolved. Unsuccessful resolution of chromatin bridges in checkpoint-deficient cells can lead to chromatin bridge breakage or tetraploidization by regression of the cleavage furrow. In turn, these outcomes can cause accumulation of DNA damage, chromothripsis, or chromosomal instability, which are associated with cancer formation or progression. We will present recent progress towards understanding the mechanisms of the abscission checkpoint and discuss its role in guarding genome integrity at the chromosome level.

Audience Take Away

- Chromatin bridges.
- Abscission checkpoint signaling.
- Mechanisms that maintain genome integrity in cytokinesis.

Biography

George Zachos completed his PhD at the University of Crete in 1997. He then received postdoctoral training in the Beatson Institute for Cancer Research, Glasgow, U.K. before moving, in 2008, to the Department of Biology, University of Crete, Heraklion, Greece as an Assistant Professor in Cell Biology. In 2015, he became Associate Professor and continues to hold this position today. Discoveries from the Zachos lab have identified mechanisms that regulate the fidelity of chromosome segregation in mitotic cell division in higher eukaryotic cells. He has published over 40 papers in leading scientific journals and his work has received ~2,000 citations.

SPEAKERS

DAY 02

5TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

16-17 SEPT

Mai-Lan Ho
Department of Radiology, Nationwide Children's Hospital, USA

Brain tumors: Imaging and molecular updates

We will discuss modern updates in imaging and classification of brain tumors. First, we will provide an overview of key World Health Organization 2021 changes in nomenclature. Next, we will summarize the utility of basic and advanced imaging techniques for brain tumors. The majority of the talk will cover case-based examples of adult and pediatric brain tumor pathologies with genotype-phenotype correlation and key diagnostic/management pearls.

Audience Take Away

- Understand World Health Organization updates in classification of brain tumors.
- Identify opportunities for utilization of basic and advanced imaging.
- Correlate molecular & radiologic characteristics of brain tumors in adult and pediatric patients.

Biography

Dr. Mai-Lan Ho is an internationally recognized radiology physician leader, scientist, and educator with experience across multiple institutions and practice models. Her clinical expertise is in genotype-phenotype correlation for malformations and syndromes of the pediatric brain, neck, and spine. Her research involves translational advanced imaging and precision medicine for systems neuroscience. She has published 95 articles, 27 grants, 4 books, 14 chapters, 74 abstracts, 18 technical reports, and nearly 300 invited talks.

Shwetima Chaudhary
Radiation oncology, All India Institute of Medical Sciences, India

A case of interstitial Brachytherapy in Carcinoma vulva with recurrence at scar site, showing results with manageable toxicity and good clinical outcome

Introduction-Vulvar cancer is a rare gynecological malignancy with incidence rates steadily increasing over the past 10 years. Despite aggressive treatment, recurrent disease is common. The goal of this case report is to give an overview of the techniques for interstitial brachytherapy for recurrent disease along with outcome and follow up after treatment.

Materials and methods- case of 70 years old female with history of post radical vulvectomy presented with recurrence after seven years at the same site. A second surgery was done at the same in the year 2022 January where she developed close margins of 0.1 mm in histopathology report. The pathological stage is pT1bN0. After complete work up as the report is suggesting close margins, she was planned for interstitial brachytherapy at the right surgical perineal area where the initial recurrence was found. Total 10 interstitial catheters were placed in 2 planes, by doing a skin marking at a distance of 1.5cm with scale covering whole of the scar site. Total dose was given 40gy in 10 fractions in twice daily dose with monitoring of skin reactions daily.

Results- On completion of radiation we observed skin reaction at 1 month and 3 months. There was no grade 2/3 toxicity, no urinary or rectal complains. Mild redness was seen post radiation which subsided with time.

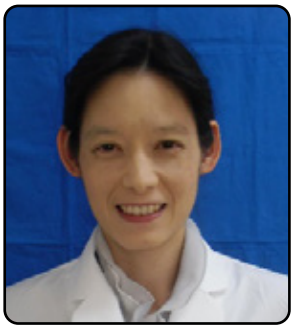
Conclusion-Interstitial brachytherapy given in a short time with a relatively small area of irradiation resulted in very good treatment tolerance. This case shows one factor that is close margin and it can be reason for recurrence later if not treated despite in stage I. Interstitial HDR brachytherapy is a safe and effective treatment modality for advanced and recurrent vulvar cancer, yielding good local control with acceptable side effects and also a point of learning about this case as we come across cases like this and also the patient was worried about the toxicity associated with radiation as she contacted many centres for radiation. But the results were excellent and post radiation minimal oedema subsided within 3 months.

Audience Take Away

- About role of interstitial brachytherapy.
- It will help to learn these techniques and gain knowledge.
- Is this research that other faculty could use to expand their research or teaching?-NO.
- This provide a practical solution to a problem that could simplify or make a designer's job more efficient.
- People in the field of radiation oncology would benefit as they can use this in their daily practice and also provides knowledge to other specialty and sub branches of oncology.

Biography

MBBS-SRMSIMS Bareilly medical college, MD (radiation oncology), Completed 2 years of senior residency (radiation oncology post MD) CCEPC (palliative care)



Yuko Harada*[1], Kyosuke Shimada, M.D.[2], Yukino Kubota, M.D.[3]

[1]Department of Cardiology, Kawasaki Municipal Ida Hospital, Japan

[2]Department of Breast Surgery, Kawasaki Municipal Ida Hospital, Japan

[3]Department of Palliative Care Medicine, Kawasaki Municipal Ida Hospital, Japan

Effectiveness of Ivabradine for Cancer Therapeutics-Related Cardiac Dysfunction (CTRCD)

As chemotherapy has advanced in recent years, 10-year survival of breast cancer patients has become as high as 79.3% in Japan. However, the prognosis of patients may be hampered by cardiotoxicity of anti-cancer treatment. It is known that Anthracyclines and Trastuzumab cause some damages to the patients' hearts even though they are very effective for breast cancer treatment.

Methods 52 breast cancer patients who underwent chemotherapy from June 2021 to June 2022 were evaluated. Three patients were diagnosed with CTRCD by criteria of European Society of Cardiology (ESC). All of the 3 patients presented with dyspnea on exertion and palpitation. Treatment for CTRCD was initiated following the guideline by American Heart Association (AHA).

Results 2 patients were treated with beta-blocker and Ivabradine, but one patient could not use beta-blockers due to asthma. All the 3 patients were treated with Ivabradine, and the symptoms of heart failure were resolved. However, 2 of them died of non-cardiac diseases.

Discussion In spite of short observation period and small patient number, Ivabradine has proven to be effective in resolving cardiac symptoms. Ivabradine could be considered for the patients who cannot tolerate beta-blockers, such as patients with hypotension, asthma, or COPD.

Audience Take Away

- The audience will learn abut Cancer Therapeutics-related Cardiac Dysfunction (CTRCD).
- The audience will learn about the new medicine to treat CTRCD.

Biography

Yuko Harada, M.D., received her M.D. degree from the Keio University School of Medicine. She is currently Vice Director of the Department of Cardiology at Kawasaki Municipal Ida Hospital. From 2018 to 2020 she was Division Head of General Internal Medicine at Yamato Tokushukai Hospital. From 2014 to 2018 she was Director of the Department of Internal Medicine at Shin-yurigaoka General Hospital. She received the Chairman's Award from the Japan Endocrinology Association for her life-saving work on thyroid storm. She has authored numerous pioneering research and medical papers in the fields of Internal Medicine, Cardiology, and Radiology.

Vladimir K. Bozhenko[1], Alexander M. Shishkin[1], Andrey N. Shkoporov[2], Yana Yu. Kiseleva[1]*, Tatyana M. Kulinich[1], Elena A. Kudinova[1], Vladimir A. Solodkiy[1]

[1] Department of molecular biology and experimental tumor therapy, Russian Scientific Center of Roentgenoradiology, Russia

[2]School of Microbiology & Department of Medicine, APC Microbiome Ireland, University College, Ireland

The CEA-specific plasmid-based CAR-T cells effectively suppress the human CEA-positive cell culture and tumor xenograft in murine model

The adaptation of CAR-therapy for a treatment of solid malignant tumors is currently considered as a highly promising opportunity for future therapies. One of the approaches in primary human T cell engineering effective commonly used for CAR gene delivery is retroviral gene transduction which can result in unpredictable transgene integration into a host genome (insertional mutagenesis). Alternatively, the electroporation of plasmid DNA or mRNA may be employed to achieve a transient immunoreceptor expression and, thereby, minimize the risk of potential complications. To realize such approach, we decide to employ plasmid DNA as more stable genetic material which does not require RNase free conditions and can be retained in cells for a long time, thereby leading to the prolonged CAR expression. Our suggested therapeutic agent, Carplasmin, is a DNA plasmid carrying the third-generation CAR directed against carcinoembryonic antigen (CEA).

The antitumor efficacy of Carplasmin was evaluated both in vitro and in vivo. First, we tested the specificity of Carplasmin-nucleofected sorted human T-lymphocytes and unsorted nucleofected lymphocytes against CEA-positive cell line HT29. Second, we assessed of the antitumor efficacy of Carplasmin, using nude mice with the intraperitoneal HCT116 (CEA-positive) xenograft as a model. Moreover we carried out the pharmacokinetic study by estimating with PCR the distribution of plasmid DNA in the blood flow of B6D2F1 mice after injection of isolated lymphocytes with Carplasmin.

In vitro experiments show the specific 50% inhibition of HT29 cells by the Carplasmin-nucleofected sorted human T-lymphocytes. T-cells electroporated with Carplasmin demonstrated in vivo a strong antitumor activity. Seven injections performed weekly resulted in a remarkable survival rate of 80% while all mice of control group (without treatment) died within that time period. The full remission was observed for 40% of treated mice. The pharmacokinetic study on lymphocytes electroporated with Carplasmin showed their ability to circulate in blood flow up to 2 weeks (with a half-life of 105±7 hours).

The results of our preclinical study demonstrate that Carplasmin may appear as a potent agent for anti-CEA therapy under conditions of repeated weekly injections.

Audience Take Away

- The advantage of plasmid-based electroporation relative to mRNA-electroporation and retroviral gene transduction.
- The audience will learn about Carplasmin – the new CEA-specific plasmid-based therapeutic agent and its antitumor activity in vitro and in vivo.
- It will be demonstrated that sorted T-cells nucleofected with Carplasmin specifically kill tumor cells whereas unsorted nucleofected and mock- nucleofected lymphocytes do it unspecifically.

- Carplasmin may appear as a potent agent for the anti-CEA therapy under conditions of repeated weekly injections.

Biography

Yana Y. Kiseleva was graduated from the Siberian State Medical University (Tomsk, Russia) and received PhD in virology and immunology in 2005 at the State Research Center of Virology and Biotechnology "Vector" (Novosibirsk region, Russia). After training at the NIH/NICHD/LCMB (Bethesda, MD, USA) as a Visiting Fellow in 2005-2007, Dr. Kiseleva worked as a Research Fellow at the Institute of Biomedical Chemistry (Moscow, Russia) and, starting 2016, at the Russian Scientific Center of Roentgenoradiology in Moscow (Russia). Her primarily research interest is in adoptive immunotherapy of cancer, mainly in CAR T-cell therapy of solid tumors.

Mamdooh Ghoneum[1], Nariman K. Badr El-Din[2], Doaa A. Ali[2] and Mai Alaa El-Dein[2], Dan Kenner[3]*

[1]Department of Otolaryngology, Drew University of Medicine and Science, U.S.A

[2]Department of Zoology, Faculty of Science, University of Mansoura, Egypt

[3]ImunoBran, Luxembourg and France

Modified Arabinoxylan from rice bran, MGN-3/Biobran, sensitizes metastatic Breast Cancer cells to Paclitaxel in vitro

There is an increased interest in alternative treatments that reduce the toxicity of chemotherapy by lowering the drug concentration, whilst maintaining potency against cancer cells. Previous studies have demonstrated that arabinoxylan from rice bran, MGN- 3/Biobran, sensitizes human breast cancer cells (BCC) to daunorubicin (DNR). In the present study, we further evaluated the ability of MGN-3 to sensitize cells to another chemotherapy agent, paclitaxel. Materials and Methods: Nonmetastatic MCF-7 (human BCC) and metastatic 4T1 (murine BCC) cells were cultured with different concentrations of paclitaxel in the presence or absence of MGN-3. Cell survival, DNA damage, and cell proliferation were examined. Results: MGN-3 increased the susceptibility of both types of cancer cells to paclitaxel by over 100-fold. Mechanistically, MGN-3 works synergistically with paclitaxel by causing DNA damage, enhancing apoptosis, and inhibiting cell proliferation in 4T1 cells. Conclusion: Our data demonstrate that MGN-3 is an effective chemosensitizer and may represent a novel adjuvant for the treatment of metastatic breast cancer.

Biography

DAN KENNER, Ph.D., L.Ac graduated in 1979 from the Meiji College of Oriental Medicine in Japan, passed the Japanese National Licensing Examination and then trained in Internships at Osaka Medical University Pain Clinic and Kinki University Medical Teaching Hospital. He is licensed to practice Oriental Medicine both in the U.S. and in Japan. He also has a Ph.D. in Naturopathic Medical Science from the First National University of Naturopathic Medical Sciences. Dr. Kenner is on the Board of Directors of the Acupuncture and Integrative Medicine College in Berkeley, California and the National Health Federation. He is author of The Whole-Body Workbook for Cancer and other titles. Since 1983, he has endeavoured to integrate the Naturopathic Medical Traditions of North America and Europe with the Traditional Medicine of East Asia.

Nicola Manfrini[1,2]*, Marilena Mancino[1,3], Annarita Miluzio[1], Stefania Oliveto[1,2], Matteo Balestra[1], Piera Calamita[1,2], Roberta Alfieri[1], Riccardo L Rossi[1], Marco Sassoè-Pognetto[4], Chiara Salio[5], Alessandro Cuomo[6], Tiziana Bonaldi[6], Marcello Manfredi[7,8,9], Emilio Marengo[7,8,10], Elia Ranzato[10], Simona Martinotti[10], Davide Cittaro[11], Giovanni Tonon[11,12], Stefano Biffo[1,2]

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[2]Department of Biological Sciences, University of Milan, Italy.
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[8]ISALIT, Italy.
[9]Department of Translation Medicine, University of Piemonte Orientale,Italy.
[10]Department of Sciences and Technological Innovation, University of Piemonte Orientale, Italy.
[11]Center for Omics Sciences, IRCCS San Raffaele Scientific Institute, Italy.
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FAM46C: a new tumor suppressor with a broad anticancer potential

FAM46C is an oncosuppressor gene which is found mutated in approximately 10% of multiple myeloma (MM) patients. Recently, we proposed a model to explain FAM46C mode of action in MM. In highly-secreting MM cells, FAM46C regulates intracellular trafficking and protein secretion dynamics, causing inhibition of autophagy and consequent accumulation of intracellular protein aggregates, an event which in turn triggers apoptosis. This effect is completely abolished by the most frequently found mutant variants of FAM46C, indicating that this activity is responsible for FAM46C oncosuppressor function. Besides describing FAM46C mode of action we also found that the expression of the wt form of FAM46C, but of none of its mutant variants, sensitizes MM cells to sphingosine kinase (SK) inhibition. Since SKs are proteins involved in ceramide metabolism, a pathway that is frequently altered in cancer cells, and SK inhibitors are currently being proposed as anti-cancer drugs, these findings underline the potential therapeutic importance of determining FAM46C mutational status in patients in order to define personalized treatment options. Emerging evidence is underlining the involvement of FAM46C in different cancer types, hence, our findings are of broad interest for the cancer research community as they might be relevant for implementing global anti-cancer strategies.

Audience Take Away

- Importance of FAM46C expression in the tumoral environment.
- Relevance of deciphering all of the intracellular pathways modulated by FAM46C for optimizing cancer treatment approaches.
- Potential use of FAM46C mutational status as a stratification method for therapeutic strategy selection/ implementation.

Biography

Dr. Nicola Manfrini studied Biotechnology at the University of Milano-Bicocca, Milan, Italy and graduated as MS in 2007. Here, he received his PhD degree in Biology in 2011. After four years of postdoctoral fellowship and a period at the Curie Institute in Paris, France, in 2015 he joined the INGM Institute in Milan, Italy. From 2021 he's Assistant Professor at the University of Milan, Italy and from 2022 he's PI at the INGM Institute of Milan, Italy.

Brandon Lucke-Wold
Department of Neurosurgery, University of Florida, USA

Innovative approaches for Breast Cancer metastasis to the Brain

Breast cancer metastasis is a continued concern for patients with recent development in our understanding of disease progression. In this paper, we highlight the pathophysiology behind breast cancer metastasis. Blood brain barrier disruption plays a critical component in progression. We then investigate the current treatment strategies and recommended guidelines. This focuses on radiation and medical management. Finally, we address the role of surgical intervention. The data is organized into tables and figures to highlight key components. Finally, we address emerging treatments and pre-clinical data. The paper will serve as a user-friendly guide for clinicians and researchers to help formulate a strategy to manage breast cancer metastasis patients sufficiently.

Biography

Brandon Lucke-Wold was born and raised in Colorado Springs, CO. He graduated magna cum laude with a BS in Neuroscience and distinction in honors from Baylor University. He completed his MD/PhD, Master's in Clinical and Translational Research, and the Global Health Track at West Virginia University School of Medicine. His research focus was on traumatic brain injury, neurosurgical simulation, and stroke. At West Virginia University, he also served as a health coach for the Diabetes Prevention and Management program in Morgantown and Charleston, WV, which significantly improved health outcomes for participants. In addition to his research and public health projects, he is a co-founder of the biotechnology company Wright-Wold Scientific, the pharmaceutical company CTE cure, and was a science advocate on Capitol Hill through the Washington Fellow's program. He has also served as president of the WVU chapters for the American Association of Pharmaceutical Scientists, Neurosurgery Interest group, and Erlenmeyer Initiative Entrepreneur group. In addition, he has served as vice president for the graduate student neuroscience interest group, Nu Rho Psi Honor Society, and medical students for global health. He was an active member of the Gold Humanism Honor Society and Alpha Omega Alpha Honor Society. He is currently a member of the Young Neurosurgeons' Committee. He is excited to join the neurosurgery residency program at University of Florida.

Yan Leyfman[1]*, Nancy Emmanuel[2], Aleksey Tentler[3], Jared Cappelli[4], Timothy K. Erick[5], Pushpa Sharma[6], Chandler H. Park[7]

[1]Icahn School of Medicine at Mount Sinai, USA
[2]University of São Paulo, Brazil
[3]Rutgers New Jersey Medical School, USA
[4]UTHSC Nashville, USA
[5]Dana Farber Cancer Center, USA
[6]Uniformed Services University of the Health Sciences, USA
[7]Norton Cancer Institute, USA

Stem cell therapies: A promising approach to tackle COVID-19 in Cancer patients

Coronavirus disease 2019 (COVID-19), a respiratory illness caused by betacoronavirus SARS-CoV-2, has broad clinical presentations ranging from asymptomatic to fatal outcomes. Studies have demonstrated that patients with severe symptoms, and uncontrolled inflammatory state have poor prognoses. Cancer patients, due to their immunocompromised status, are at an increased risk of severe SARS-CoV-2 infection. Given the diverse clinical presentations, we developed a model to explain SARS-CoV-2's pathogenesis and interplay with cancer. Since SARS-CoV-2 causes multi-organ dysfunction through IL-6-mediated inflammation and hypoxia, while malignancy causes apoptosis through hypoxia-induced cellular metabolic alterations, we propose a mechanism by which both conditions resulted in IL-6 upregulation causing increased cytokine release and systemic injury with clinical support.

In this model, infection with SARS-CoV-2 and malignancy results in increased IL-6 production leading to enhanced systemic injury as compared to either alone. Currently, there are limited effective therapeutic interventions against severe SARS-CoV-2. Due to its complex nature, we propose the use of combination therapies that can control the systemic inflammation induced by this condition, while halting viral replication. One approach is the use of a stem cell therapy that has yielded promising efficacy in COVID-19 patients with severe disease. This therapy, mesenchymal stem cells, possesses regenerative, antiviral and immunomodulatory properties that can inhibit viral replication, while dampening the cytokine response with resulting systemic inflammation and injury. Clinically, it has demonstrated over a 90% overall survival and 100% survival in patients younger than 85 years old within a month after treatment with results holding steady for 6 months. Thus, cancer patients can quickly contain SARS-CoV-2 with limited interruptions to their treatment schedule.

Looking forward, we foresee further follow-up studies on this therapy, including in combination with others, that can target disease pathogenesis at multiple steps within the pathway to hinder direct viral injury, suppress IL-6 release, and dampen systemic inflammation that can better thwart the virus's heterogeneity and mutational adaptations.

Audience Take Away

- This presentation will feature our cohesive models for the mechanism of action for SARS-CoV-2, the first synergistic paradigm between the flu and SARS-CoV-2 termed as "COVI-Flu", and the first proposed mechanism to showcase the interplay between cancer & COVID-19.
- This presentation will explain and highlight the efficacy of a mesenchymal stem cells—a promising cellular therapy that in clinical trials against COVID-19 has yielding promising efficacy with minimal side effects.

- The results from this presentation will serve to increase interest in the field of stem cell therapies and their promising potential to treat disease.
- This presentation will drive great investment into mesenchymal stem cell research to further fine tune their efficacy to treat more disease.

Biography

Dr. Yan Leyfman, MD, has been recognized as one of the top international researchers in oncology by ASH and ASCO. During the COVID-19 pandemic, he was recruited as the Director of the Immunology Division of the Global COVID-19 Taskforce, which produced one of the first mechanisms for SARS-CoV-2 and COVI-Flu along with therapeutic interventions for both. In 2021, Dr. Leyfman presented the first mechanism to explain the interplay between cancer and COVID-19 at the 2021 ASCO Annual Meeting. His work has been published as the cover article in the journal, Shock, and in the textbook, Insights on a Post-COVID World.

Suhas Sampat Kharat*, Xia Ding, Divya Swaminathan, Shyam Sharan
Mouse Cancer Genetics Program, National Cancer Institute, USA

Epigenetic marker on DNA regulates PARP inhibitor resistance

Poly (ADP-ribose) polymerase inhibitor (PARPi) -induced synthetic lethality of BRCA-deficient cells is being utilized to treat breast and ovarian tumors. However, emergence of resistance to PARPi remains a major concern and understanding resistance mechanisms is of utmost clinical importance. In addition, it provides mechanistic insights into biological processes that are affected by BRCA-deficiency. To identify new regulators of PARPi-resistance in BRCA2-mutant cells, we performed a genome wide siRNA screen in mouse embryonic stem cells (mESCs). We found *Ten Eleven Translocation 2 (TET2)* loss contributes to resistance to PARPi (Olaparib). Validation of knockdown of TET family of proteins in BRCA2 deficient cells exhibited chemoresistance to not only olaparib but to other PARPi such as Veliparib, Talazoparib and platinum-based drugs such as cisplatin. TET2 is a metabolic enzyme that oxidizes 5-methylcytosine (5mC) to 5-hydroxymethycytosine (5hmC), 5-formylcytosine (5fC) and 5-carboxylcytosine (5CaC) during DNA demethylation. Interestingly, we found that TET2 knockdown protects stalled replication forks in BRCA2 deficient cells. Replication fork protection is attributed to the reduction in 5hmC levels on the chromatin and not to changes in the expression of proteins associated with replication fork integrity. Proximity ligation assay revealed that 5hmC is localized on replication fork. Furthermore, we show that increase in 5hmC due to Vitamin C can induce degradation of stalled replication forks and cause genomic instability. We also demonstrate that Base Excision Repair associated apurinic/apyrimidinic endonuclease, APE1, is responsible for degradation of 5hmC, 5fC and 5CaC containing replication fork. Vitamin C is cofactor for TET proteins. Combined treatment of Vitamin C and Olaparib resensitized PARP inhibitor resistance cells to Olaparib. Our findings reveal a novel role for 5hmC, an epigenetic mark on the DNA, in maintaining the integrity of stalled RF and PARP inhibitor sensitivity. Future experiments are focused on increasing 5hmC levels to resensitize chemoresistant BRCA2 tumors.

Audience Take Away

- Novel mechanism of chemoresistance in BRCA2 deficient cells.
- Role of 5hmC and Vitamin C in PARP inhibitor resistance.
- This study can be used to redesign PARP inhibitor therapy to enhance its efficacy and overcome problem of chemoresistance.

Biography

Dr. Kharat obtained his B.Sc. in Biotechnology from Pune University, India and M.Sc. in Biotechnology from Madurai Kamaraj University, India. He completed his Ph.D. studies under the supervision of Dr. Sagar Sengupta at National Institute of Immunology, New Delhi in 2015. In 2016, Dr. Kharat joined the laboratory of Dr. Shyam Sharan as a postdoctoral fellow in the Mouse Cancer Genetics Program at National Cancer Institute, Frederick. His current research is focused on understanding chemoresistance mechanisms in BRCA2 deficient cells. In 2020 Dr. Kharat received Fellow Award for Research Excellence award (FARE) from NIH for his postdoctoral research.

Mehrasa Nikandish
University of Georgia, Georgia

Cancer drugs market

Presentation will be about cancer drugs market which is popular for the treatment.

Audience Take Away

- The rise of the **cancer/oncology market** of drugs.
- Comparison between the market of cancer drugs in different countries.
- Different kinds of treatment (Immunotherapy and Hormone therapy).

Biography

Mehrasa Nikandish, pharmacy bachelor degree at the university of Georgia, which is located in Georgia, Tbilisi and Graduated in 2022. I have some experiences to do research and review articles and also participate in national which was held by university and international conferences which was held virtually during covid-19 time. It is my second time that I am participating as an oral presenter in magnus conferences. I am also selected as the best presenter in Georgian conferences which was held by university of Georgia. And also publication of two full review articles by ESJ journal and several abstracts by cacasus journal of health science.

Anyou Wang
University of Memphis, USA

Noncoding RNAs work as the most important endogenous regulators for all Cancers

All cancers share an endogenous regulatory realm. Understanding this regulatory realm helps to comprehend the universal mechanism of all cancers; however, the conventional approach fails to uncover it due to heterogeneous biological data. This study reveals the endogenous regulatory network of all cancers by identifying endogenous regulatory interactions from big heterogeneous RNA_seq data and unearths the most important endogenous regulators for the cancerous realm. Noncoding RNAs predominate the entire cancerous realm and pseudogenes serve as the most important mediators cis-regulating their neighbors, in which they primarily mediate their targets within 1 million base pairs but they rarely target their cognates with complementary sequences as thought. Clinical data also validate noncoding RNAs as the deadliest universal regulators for all cancers. Furthermore, noncoding RNA biomarkers can discriminate all cancer types from normal tissues with high accuracy. Therefore, noncoding RNAs, instead of proteins, as traditionally thought, function as endogenous rulers that crucially rule the regulatory realm of all cancers.

Biography

Anyou Wang received his PhD from University of California, Riverside. His research interest is in computational biology, artificial intelligence and big data. Dr. Wang develops computational algorithms to catch up the big pictures from massive data and to understand the fundamental principles of biology (combai.org). He computed petabyte level data and revealed the distinctive functional regime of endogenous lncRNAs in dark regions of human genome and unearthed that noncoding RNAs endogenously rule the cancerous regulatory realm while proteins govern the normal. Recently, Dr. Wang also developed a novel alignment-free system integrating Fréchet distance and artificial recurrent neural network to reveal the evolutionary trajectory and origin of SARS-CoV-2 from more than two millions of genome sequences.

Ibrahim Yaseen[1]*, Pete Monk[2], Lynda Partridge[3]

[1]Department of Laser and Spectroscopy, University of Al-Hamdaniya, Al-Hamdaniya, Iraq
[2]Department of Infection, Immunity and Cardiovascular Disease, University of Sheffield, UK
[3]School of Biosciences, University of Sheffield, UK

The Tetraspanin Tspan2 membrane protein: A potential target for Cancer therapy

Tetraspanin proteins cross the membrane four times and span the cell surface to form two extracellular loops (EC1 and EC2) and one small intracellular loop, and typically short cytoplasmic N- and C-termini. They are involved in many biological pathways such as viral, bacterial, and parasite infection, immune response activation, cell migration and adhesion, sperm-egg fusion, progression in tumour biology, and metastasis suppression. Tspan2, a member of the tetraspanin family, may play a role in nervous system development through its signaling association in the early stages of oligodendrocytes' terminal differentiation into myelin-forming glia. Previous studies have reported the involvement of Tspan2 in the cellular motility and invasiveness of human lung cancer cells. However, the tissue-wide expression of Tspan2 protein and the associated biological activities are not yet fully understood. Also, the generation of monoclonal antibodies (mAbs) against the native tetraspanin proteins is challenging due to the sequence conservation and the fact that antibodies raised to peptides or recombinant proteins fail to recognise the native protein. Therefore, we aimed to develop mAbs are able to bind to the native form of the protein, in which mouse myeloma cells (NS0) transfected with the full-length Tspan2 were used for the immunisation. In addition to the generation of novel mAbs against the native form of the transmembrane protein, the current study explored the localisation of Tspan2 in the human lung adenocarcinoma cell line. Tspan2 blocking via anti-Tspan2 mAb showed to reduce cancer cell adhesion.

Audience Take Away

- The audience will be introduced to the newly discovered tetraspanin protein (Tspan2) and they can learn how important is the tetraspanin proteins' role in cancer biology.
- The audience will learn how new techniques in monoclonal antibody development can raise antibodies that are capable to recognises the native form of the protein, which in turn shall develop their experience in the pharmaceutical industries.
- The role of Tspan2 in cancer biology is not fully understood, and researchers might develop novel ways to research the possibility of recruiting this protein as a distinctive biomarker.

Biography

Dr Ibrahim Yaseen studied Molecular Biology and Biotechnology at the University of Sheffield, the UK, and graduated as PhD in 2017. He joined the research group of Drs Lynda Partridge and Pete Monk at the School of Biosciences and the Medical School respectively. Dr Yaseen's research focused on the transmembrane proteins tetraspanin family and their biological activities in health and diseases.

Ankit Naik[1]*,Nidhi Dalpatraj[1], Noopur Thakur[1]

[1]Biological and Life Sciences, School of Arts and Sciences, Ahmedabad University, India

Global gene expression regulation by TGFβ in Prostate Cancer through H3K4me3and H3K9me3 mark

Epigenetic alterations play an important part in carcinogenesis. Different biological responses, including cell proliferation, migration, apoptosis, invasion, and senescence, are affected by epigenetic alterations in cancer. In addition, growth factors, such as transforming growth factor-beta (TGFβ), are essential regulators of tumorigenesis. The molecular mechanisms of action of TGFβ are understood mainly through the canonical and non- canonical signal transduction pathways. Our understanding of the mechanisms that establish transient TGFβ stimulation into stable gene expression patterns remains incomplete. Epigenetic marks like Histone H3 modifications are directly linked with gene expression and play an essential role in tumorigenesis. However, the effects of TGFβ signaling on the genome wide H3K4me3 and H3K9me3 landscape remain unknown. In this study, we performed chromatin immunoprecipitation-sequencing (ChIP-Seq) to identify the genome-wide regions that undergo changes in H3K4me3 and H3K9me3 occupancy in response to TGFβ stimulation in cancer. We also show that TGFβ stimulation can induce acute epigenetic changes through the modulation of H3K4me3 and H3K9me3 marks at genes belonging tospecial functional categories in prostate cancer. TGFβ induces the H3K4me3 mark on its ligands like TGFβ, GDF1, INHBB, GDF3, GDF6, and BMP5, suggesting a positive feedback loop. Most genes were involved in the positive regulation of transcription from the RNA polymerase II promoter in response to TGFβ. Other functional categories were intracellular protein transport, brain development, EMT, angiogenesis, antigen processing, antigen presentation via MHC class II, lipid transport, embryo development, histone H4 acetylation, positive regulation of cell cycle arrest,and genes involved in mitotic G2 DNA damage checkpoints. On the other side, H3K9me3 occupancy increases in intronic regions after short-term (6h) TGFβ stimulation and in distal intergenic regions during long-term stimulation (24h). We also found evidence for a possible association of SLC transporters with the H3K9me3 mark in the presenceof TGFβ during tumorigenesis. The epigenetic mechanisms-mediated regulation of gene expression by TGFβ was concentrated at promoters rich in SRY and FOXJ3 binding sites. Our results point toward a positive association between the oncogenic function of TGFβ and the H3K9me3 mark and provide a context for the role of H3K9me3 in TGFβ-induced cell migration and cell adhesion. Interestingly, these functions of TGFβ through H3K9me3 mark regulation seem to depend on transcriptional activation in contrast to the conventionally known repressive nature of H3K9me3. Interestingly, these functions of TGFβ through H3K9me3 mark regulation seem to depend on transcriptional activation in contrast to the conventionally known repressive nature of H3K9me3. Our results link TGFβ stimulation to acute changes in gene expression through an epigenetic mechanism. These findings have broader implications on epigenetic bases of acute gene expression changes caused by growth factor stimulation.

Audience Take Away

- The Audience will be getting a detailed overview of how TGFβ can modulate global gene expression during cancer progression through specific Histone marks i.e., H3K4me3 and H3K9me3. Also, the audience will be presented will be presented with some novel information that opens up new avenues in the field of cancer epigenetics.

Biography

Mr. Ankit studied Biotechnology at the Nirma University and graduated as M.Sc. in 2017. He then joined the Lab of Prof Noopur Thakur at Ahmedabad University as Doctoral Student. He was awarded the INSPIRE fellowship by Department of Science and Technology, Govt. of India, for pursuing doctoral studies. He has published two research papers as first author in International Peer-review Journals.

Roque Gabriel Wiseman Pinto
Professor & Head, Department of Pathology Goa Medical College, India

Recent advances in pediatric renal tumors

In the pediatric age group, Wilms Tumor (Nephro blastoma), is the commonest (85%). The others being Congenital Mesoblastic Nephroma, Clear Cell Sarcoma and Rhabdoid Tumor.

Wilms Tumor is a Malignant Embryonal Tumor arising from the Nephrogenic blastema and has 3 components blastema, epithelial and stromal component,(triphasic), sometime it is biphasic and monophasic. The prognosis and management is done by 2 systems.

1. SIOP (International Society of Pediatric Oncology) and
2. COG (Children Oncology Group)

The Genetic markers are WT1 gene at chromosome 11p13, WT2 gene at chromosome 11p15, PAX6, Familial WT1, Familial WT2 and LOH (Loss of Heterozygosity) The Pathological features, like Gross, Microscopy, Clinical features etc will be discussed.

The prognosis depends on the Tumor stage at Diagnosis, Histology, Favourable verses unfavourable histology (diffuse anaplasia), age, LOH at chromosome 1p and 16q.

The other tumors are Congenital Mesoblastic Nephroma which presents at birth or below 6 months of age, has ETV6-NTRK3 gene fusion, Clear Cell Sarcoma (poor prognosis) and BCOR gene, and the Rhabdoid Tumor which has loss of SMARCB1 (1N1 – deficient tumor)

Biography

Dr.Pinto completed MBBS & MD Pathology from Goa Medical College & University of Bombay standing First in the University. He also passed National Board of Examination (DNB) in Pathology. He joined the Pathology Department of Goa Medical College on 1st February 1984 as a Demonstrator and has been promoted and served the Department as Lecturer, Assistant Professor, Associate Professor and Professor & Head and Dean Goa University. Currently he is the Professor and Head of Pathology Department,Goa Medical College. He has 230 Publications and has been invited as Faculty for International Conferences, CMEs and workshops all over the World. He is the Editor in Chief of Todays Clinician and Member of Editorial Board of many Journals. He has organized many International Conferences and CMEs , in Goa and has held many prestigious positions in Academic bodies. Like President - Indian Academy of Cytologists & President - Asian Society of Cytopathology, Chairman - International Affairs Committee, IAC,Chairman , Board of Studies, Goa University, Executive Council Member Goa University, Academic Council Member Goa University, Chairman Multi Disciplinary Research Unit(MRU), Professor In charge Medical Education Unit, Goa Medical College.

Dipika Bumb
Department of Public Health, M.S. Ramaiah University of Life and Allied Sciences, India

Targeting health disparities in oral Cancer outcomes through technology in LMIC's- A public health priority

Cancers of the lip, oral cavity, hypopharynx, oropharynx and larynx are known collectively as mouth andoral cancers. Of these, cancers of the lip and oral cavity are the most common, with more than 377,700 cases worldwide in 2020. (GLOBOCAN). In India, the common leading cancer sites are breast, lung, mouth, cervix uteri, and tongue. (NCRP2020).

Tobacco has been the prime cause of oral cancer, and is identified as a disparity with a strong relation to socio- economic status of an individual. Health disparities is defined as, "preventable differences in the burden of disease,injury, violence, or opportunities to achieve optimal health that are experienced by socially disadvantaged populations." (CDC). Individuals of low-SE status experience disproportionate & unique social and environmental challenges that contributes to tobacco use like residence, occupation, education, levels of perceived stress, social discrimination etc., therefore making them more susceptible to develop oral cancer at some stage of their life. This high-risk population, also has poor access to screening for potentially malignant disorders, diagnosis and treatment. Together these factors, lead to delayed diagnosis with low survival outcomes & severe anatomical defects associated with poor physical & psychological quality of life. Studies have shown that there is enough awareness about destructive effects of tobacco use, however little is known about the early lesions, signs and symptoms, the impact of continued tobacco use (smoking & smokeless) during treatment of oral cancer. The NPCDCS program India, has revisedguidelines with inclusion of screening for oral breast and cervical cancers but due to the paucity of trained health workers at the Health and Wellness centers, PHC & CHC levels, creates a major setback and is a hurdle for professional, accessible care to this segment of population. This leads to reluctancy in reporting, ultimately people reaching thehospitals to seek medical care in advanced stages. Newer technologies like AI, telemedicine, m-health, focused onevidence-based approaches around health promotion, communication, capacity building & standardized data collection can prove to be of great significance in addressing the above-mentioned disparities leading to systems strengthening in low resource settings.

Audience Take Away

- Understand health disparities & inequalities in oral cancer in LMIC's.
- Different areas & strategies to develop an evidence-based intervention like usability of quit smoking programs on smartphones in low resource settings etc.
- To mention the limitation or challenges of technology-based solutions.

Biography

Dr. Dipika Bumb, studied Oral Medicine and Radio-diagnosis at the University of Rajasthan, India. She then worked with Indian Cancer Society as an Oral cancer screening expert & research officer. She led many projects involving e- health records, teach the trainee, screening programs in migrated populations & gender resource centers. She then joinedPublic Health Foundation of India in NCI Planning grant on establishing Centers of Research Excellence in cancer. Shealso served a s a scientist at the National Cancer registry program, ICMR, Bengaluru. She has 16 publications and has been listed in the Marquis Who's Who biography in 2015. She has received IACA fellowship Award to pursue CPFP.

Samiah Shahid[1]*, Maryam Javaid[1], Wajeehah Shahid[2]

[1]Institute of Molecular Biology and Biotechnology, The University of Lahore, Pakistan.

[2]Department of Physics, The University of Lahore, Pakistan.

Expression of miR-146a-5p and *PBX2* gene in xenograft animal model as circulating markers for early diagnosis of Acute Lymphoblastic Leukemia

MicroRNAs have shown its role in cell proliferation, apoptosis and pathogenesis of acute lymphoblastic leukemia (ALL). Current diagnosis of ALL comprises invasive and inconvenient procedures including bone marrow biopsy. In spite of recent advancements in the treatment of ALL, still there is risk of relapse. MiR-146a-50 and its target gene *PBX2* has been reported to have a potential role in ALL. So the present study was designed to evaluate the expression of miR-146a-5p and *PBX2* gene in xenograft rabbit model that can be utilized as circulating markers for early diagnosis of ALL. ALL was induced in rabbit model using xenograft method. Whole blood (5 mL) was collected in EDTA tubes from the ALL-induced rabbits and healthy rabbits after approval from animal ethical committee of The University of Lahore. Plasma was isolated and the total RNA including miRNA was extracted using miRVana isolation kit from Thermofischer Scientific (USA). The concentration and purity of total RNA including miRNA was determined by NanoDrop. Reverse transcription of total RNA including miRNA was performed by using SYBR green kit from Thermofischer Scientific (USA) as per to manufacturer's protocol. The quantitative real-time PCR was performed to check the expression of miR-146a-5p in ALL induced and healthy control rabbit samples. Normalized fold expression was calculated by $2^{-\Delta\Delta Ct}$ method using miR-16 as normalizer. The experiment was performed in duplicate and statistical significance of the results was analyzed using SPSS and Graphpad Prism software. The results showed significant up-regulation of circulating miR-146a-5p and *PBX2* gene with a mean fold expression of 102±20.79 and 9.18±0.160 respectively, in ALL induced rabbits as compared to healthy controls (P<0.05). miR-146a-5p and *PBX2* gene expression levels may be utilized as circulating markers for early diagnosis of ALL. In future, targeting miR-146a-5p and its target gene *PBX2* may be utilized as therapeutic target of ALL.

Audience Take Away

- The work done in the present study will help audience to learn about recent advancements for early and non-invasive diagnosis and treatment of cancer particularly acute lymphoblastic leukemia (ALL) that is a type of Blood cancer.
- As micro RNAs are emerging as circulating biomarkers, so this study may open a new horizon for researchers, scientists and medical professionals to use such diagnostic tools for cancer.
- Future research may be done to find microRNA based therapeutic target for the treatment of cancer.
- The current research will help researchers, scientists as well as medical professionals to implement microRNA based diagnostic procedures for early diagnosis of ALL and other cancers as well.
- The study provides solution to replace invasive procedures like biopsy to non-invasive procedures as mentioned in this study, circulating microRNA. Through this research, blood-based diagnosis of cancer may be possible.

Biography

Dr. Samiah Shahid studied Masters in Biochemistry at University of Agriculture Faisalabad in 2009 with gold medal. She then joined Biopharmaceutical and biomarker discovery lab at School of Biochemistry and Biotechnology, University of the Punjab and received her Ph.D degree with research on MicroRNA Profiling for early diagnosis, prognosis and treatment of cancer. She has a number of Publications in the field of cancer. She has >10 years of teaching and research experience in medical colleges/research institutes. She is now of Associate Professor at Institute of Molecular Biology and Biotechnology/Center for Research in Molecular Medicine, The University of Lahore, Pakistan.

Ali Mamivand[1*], Shiva Bayat[1], Abolfazl Maghrouni[1], Sasan Shabani[1], Alireza Khoshnevisan [2], Hiva Saffar[3], Mina Tabrizi[1]

[1]Department of Medical Genetics, School of Medicine, Tehran University of Medical Sciences, Iran
[2]Department of Neurosurgery, Shariati Hospital, Tehran University of Medical Sciences, Iran
[3]Department of Pathology, Shariati Hospital, Tehran University of Medical Sciences, Iran

Data mining of bulk and single-cell RNA sequencing introduces OBI1-AS1 as an astrocyte marker with possible role in Glioma recurrence and progression

Long non-coding RNAs (lncRNAs) are widely known for their various functions in cancer from tumor initiation to tumor progression and metastasis. Gliomas are the most prevalent primary forms of brain tumor, classified into grades I to IV according to their malignant histological features with grade IV, also known as Glioblastoma Multiforme (GBM), displaying the highest level of malignancy. Thus, the search for Differentially Expressed LncRNAs (DELncRNAs) in GBM versus Low-Grade Glioma to uncover new insights into the molecular mechanisms of glioma progression have intensified. Bulk RNA sequencing pinpointed decreased expression of OBI1-AS1 in GBM compared to Low Grade Glioma (LGG) samples. Subsequent single nuclei RNA sequencing revealed OBI1-AS1 to be a super-exclusive astrocyte marker with AUC=0.99 and the potential to fully differentiate astrocytes from other brain cells. Additional supplementary bioinformatics analysis exhibited OBI1-AS1 role in synaptic signal transduction and glutamatergic signaling. In addition, ChIP-Seq data was analyzed to explore transcription factors that can regulate OBI1-AS1 expression in neural cells. Results of Hi-C, methylation and ChIP-Seq analysis strongly suggest methylation of the CTCF binding site serving a central role in regulation of OBI1-AS1 expression via managing chromatin interactions. Our study indicated that lncRNAs, like OBI1-AS1, could be extremely precise in identifying the astrocyte cluster in the single-cell transcriptome and demonstrating superiority to well-established astrocyte markers such as GFAP, S100B, ALDH1L1, and AQP4.

Audience Take Away

- In the current study, we show that DNA hypermethylation in orchestration with CTCF can increase gene expression by regulating the chromatin territories.
- Our study reveals the implication of lncRNAs like OBI1-AS1 in the recurrence of Glioma.
- Our single-cell RNA sequencing analysis revealed that *OBI1-AS1* could be a super exclusive marker for astrocytes. That is an attention-worthy finding which introduces OBI1-AS1 as a candidate marker for distinction of astrocytes from other brain cell types during cell annotation in the single cell analysis process.

Biography

Mr. Mamivand studied human genetics at the Tehran university of medical science (TUMS), Iran and graduated as MS in 2021. He joined the research group of Dr. Tabrizi at the TUMS. During his master, he studied brain tumors, especially glioma, from the perspective of cancer epigenomics.

Mahmoud Maher Abdelnaby Alrahawy
East Suffolk and North Essex NHS Foundation Trust, United Kingdom

The role of texture analysis of MRI in prediction of local recurrence and distant metastasis in locally advanced Rectal Cancer

Aim: Locally advanced rectal cancer (LARC) is treated by neoadjuvant chemoradiotherapy (NCRT) followed by surgery after restaging with magnetic resonance imaging (MRI). Texture analysis (TA) is an imaging biomarker that could assess MRIs heterogeneity by measuring grey-level intensities distribution. This study hypothesizes that TA can predict local recurrence and distant metastasis.

Method: This is a retrospective analysis of LARC patients after NCRT. From the posttreatment MRI scans, the tumor's Region of interest (ROI) was determined on T2 MRI images. Six texture parameters were systematically extracted and were examined to predict local recurrence and distant metastases through Kaplan-Meier survival curves and log-rank tests.

Results: From 113 patients with LARC, two texture parameters significantly predicted local recurrence: Entropy (p=0.033) and mean of positive pixels (MPP) (p=0.045). Meanwhile, five parameters predicted distant metastases: SD(p=0.015), entropy(p=0.017), MPP(p=0.005), skewness (p=0.046), and Kurtosis (P=0.019). Kaplan-Meier Log rank test showed that entropy and skewness independently predicted distant metastases.

Conclusions: MRI textural features are potentially significant imaging biomarkers in predicting local recurrence and distant metastases in LARC treated with NCRT.

Key Statement: This study indicates that textural parameters could predict local recurrence and liver metastasis in LARC patients. This could formulate an algorithmic model to personalize the treatment of cancer patients by using a non-invasive imaging biomarker. However, more clinical, histological and genomic correlations is still required in the future research.

Biography

Mr Alrahawy studied medicine at the Menoufia University in Egypt and graduated with a Bachelor's degree in medicine in 2010 with an excellent degree of honour. He then joined a general surgery residency at Menoufia University hospital and finished his Master's degree in general surgery in 2016. In 2018, he travelled to the UK through a joint supervision programme) as a research fellow at the ICENI centre, in Colchester. In 2022, He was granted his MD in from Menoufia University and now he is obtaining the position of lecturer of General Surgery at Menoufia University.

Nadia Senhaji [1,2]*, Asmae Squalli Houssaini [2] , Salma Lamrabet [2] , and Sanae Bennis [2]

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Circulating biomarkers in Glioblastoma: A valuable tool to improve patient care

Despite important advances in the management of tumors in recent decades, glioblastoma (GBM) remains one of the deadliest human cancers. In fact, GBM presents the lowest median observed survival of all primary malignant brain and other CNS tumors (8 months). This poor prognosis is mainly caused by therapeutic resistance and recurrence after surgical removal. Current therapeutic approaches for GBM combine surgery, radiotherapy, and chemotherapy. Still, even with the use of Temozolomide, the standard chemotherapeutic drug, GBM patients show a low median survival of ~15 months. While tissue biopsy remains the standard procedure commonly used for the histological characterization of GBM, liquid biopsy has recently emerged as a promising way to improve patient care in terms of initial diagnosis, relapse, and choice of appropriate treatments. Indeed, liquid biopsy gives the possibility of taking repetitive and non-invasive samples, which allows real-time monitoring of the patient during treatment. Blood, serum, and cerebrospinal fluid carry biomarkers such as circulating tumor cells, cell-free nucleotides, extracellular vesicles, and circulating proteins that are linked to diagnosis and/or prognosis. Moreover, some of these particles might be used to detect therapeutic resistance or identify tumor recurrence. However, to date, no circulating biomarkers for managing GBM have been clinically validated. As each biomarker has advantages and disadvantages, a combination of biomarkers could be valuable to obtain diagnostic and prognostic information in a non-invasive method. Nonetheless, further studies with larger cohorts are needed in order to increase specificity and sensitivity, and to promote future clinical applications.

Audience Take Away

- Shed light on the latest technologies used in the detection of circulating biomarkers.
- Compare the advantages and disadvantages of the different techniques.
- Emphasize the usefulness of these means for better management of patients with Glioblastoma.

Biography

Pr. Nadia Senhaji obtained her PhD degree in 2016 in the field of molecular biology and oncogenetics. During her years of thesis preparation, she was able to integrate the Laboratory of Biomedical and Translational Research, Faculty of Medicine, Pharmacy and Dental Medicine of Fez, Sidi Mohamed Ben Abdellah University. She became assistant professor in genetics and molecular biology from 2020, at the Faculty of Sciences of Meknes, Moulay Ismail University in Morocco. Since then, she has continued her research work specifically in the oncogenetics of brain tumours. She has published dozens of articles in indexed scientific journals.

POSTERS

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16-17 SEPT

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Associated factors with post-traumatic growth among spouses of women diagnosed with Gynecological Cancer: A study from China

Aim: The aim of this study was to explore the factors that are associated with posttraumatic growth (PTG) among spouses of women diagnosed with gynecological cancer (GC).

Design: A cross-sectional descriptive study.

Methods: A convenience sample of 312 spouses of women diagnosed with gynecological cancer was recruited from two comprehensive hospitals in China, from March 2018 to March 2020. Demographic characteristics, cancer-related characteristics, posttraumatic growth, perceived social support and coping were assessed using self-reported questionnaires. Descriptive statistics and multiple linear regression analysis were performed.

Results: The mean score of post-traumatic growth was 46.7 (standard deviation=16.7). The associated factors of post-traumatic growth were spouses' age, perceived social support, problem-focused coping, dysfunctional coping and cancer treatment received by partners, which accounted for 34% of total post-traumatic growth score.

Conclusion: Spouses' age, perceived social support, problem-focused coping, dysfunctional coping and cancer treatment received by partners were associated with PTG among spouses of women diagnosed with GC. Importantly, the findings in this study suggest that positive support-seeking and perceiving more social support from family and others, and using dysfunctional coping, could be intervention strategies to facilitate spouses' PTG, particularly in young spouses and spouses of women who receive surgery only. Specifically, these findings are meaningful for the development of family-based interventions, which could focus on perceived social support and coping to help spouses to foster more PTG. For example, spouses who have low levels of perceived support from family may benefit from disclosure between family members and promote/maintain intimacy relationship.

Relevance to clinical practice: The findings from this study provide a deeper understanding of PTG and its associated factors in a Chinese cultural context, which has valuable implications for clinical practice. First, the positive association between perceived social support and PTG suggests that health professionals should be aware of the need for increased attention and should provide appropriate support to spouses, such as developing a support intervention based on the spouses' care needs Perceived support from family was most common in spouses, suggesting that the family members should be the first consideration for spouses to increase their PTG in a Chinese cultural context. For example, encouraging spouses to communicate with family members, building and maintain meaningful relationships with family members, and ensuring that spouses make use of the existing family support available to them. Second, the positive association between dysfunctional coping and PTG indicates that health professionals need to evaluate spouses' coping and help spouses to choose the appropriate coping behaviors via nursing intervention. In particular, according to the recommendation proposed by Tedeschi et al. (2018), a nursing intervention comprising coping and social support should be integrated into existing cancer care practice because the early phase of cancer diagnosis and its treatment offer important moments for promoting health behavior change and PTG. Third, given that spouses' age and partners' cancer treatment are associated factors of spouses' PTG, health professionals should provide effective care to younger spouses and spouses of women who have been prescribed with surgery only.

Audience Take Away

- This study is possibly the first study to explore the association between PTG, perceived social support and coping among spouses of women diagnosed with GC. The findings from the present study indicated that spouses with high levels of perceived social support and dysfunctional coping showed high levels of PTG, while spouses of younger age and spouses of partners who had been given surgery only showed low levels of PTG.
- In this study, the subscale score of support from family was significantly higher than support from statistically significant other and support from friends. This finding indicates that perceived support from family may be more helpful in supporting spouses to experience more PTG during their caring experience.
- Particularly in young spouses and spouses of women who receive surgery only, positive support-seeking and perceiving more social support from family and others, and using dysfunctional coping, could be intervention strategies to facilitate spouses' PTG.
- The positive association between perceived social support and PTG suggests that health professionals should be aware of the need for increased attention and should provide appropriate support to spouses, such as developing a support intervention based on the spouses' care needs.

Biography

Prof. Lihua Zhou graduated as MS in 2008 and studied as a PhD student from 2017. During her PhD study with the guidance of all supervisors (i.e., Jingfang Hong, Anhui Medical University; Maria Browall, Jönköping University; Maria Henricson, Uppsala University; Margaretha Stenmarker, University of Gothenburg Sahlgrenska Academy). She joined the ADULT research group in the School of Health and Welfare in Jönköping University. The title of her PhD thesis is 'Personal growth and its associated factors among women diagnosed with gynecological cancer, spouses and registered nurses: A study from China'. This presentation is a part of her PhD thesis. She will defense her thesis in 202306.

Participants List

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