Track 9(i) 9(ii) 9(iii) 9(iv) 9(v)

9(i): Breast cancer
9(ii): Liver, Prostate & Kidney Cancer
9(iii): Blood, Lung & Leukemia Cancer
9(iv): Bone & Thyroid Cancer
9(v): Gastrointestinal & Colorectal Cancer

Session Chair

Dr. Samir A. Farghaly

The Medical College of Cornell University, USA

Session Co-Chair

Dr. Eldad Zacksenhaus

University Health Network, Canada

Session Introduction

Title: **Identifying and targeting tumor initiating cells (TICs) in mouse models of breast cancer**

Dr. Eldad Zacksenhaus, University Health Network, Canada

Title: **Susceptibility alleles of breast cancer in high, moderate and low penetrance genes in a South American population**

Dr. Lilian Jara, School of Medicine-University of Chile, Chile

Title: **Pharmacogenomics predictions for breast cancer treatments´ efficacy and toxicity**

Dr. Hugo A Barrera Saldaña, University of Nuevo León, México

Title: **A novel feedback loop with therapeutic implications in estrogen receptor-negative breast cancer**

Dr. Ali Naderi, The University of Queensland, Australia

Title: **Molecular classification of breast cancer and role of immunohistochemistry for detection of cell types that predicts response to chemotherapy with Santinib (P53)**

Dr. M. H. Bukhari, King Edward Medical University, Pakistan

Title: **Combining biologics and cytotoxics in the treatment of inoperable cholangiocarcinoma**

Dr. Lars Henrik Jensen, Vejle Hospital and University of Southern Denmark, Denmark

Title: **Stability analysis on liver cancer related miRNA in serum**

Dr. Xian-Feng Ding, Zhejiang Sci-Tech University, China

Track 9(i) 9(ii) 9(iii) 9(iv) 9(v)

Title: **The status of anti-metastatic gene therapy in Patients with advanced epithelial ovarian cancer**

Dr. Samir A. Farghaly, The Medical College of Cornell University, USA

Title: **Detection and quantification of circulating Melanoma cells in patients with cutaneous malignant Melanoma**

Dr. Mel Ziman, Edith Cowan University, Western Australia

Title: **A high prevalence of metabolic syndrome in a group of colorectal patient's pre surgery– A pilot study**

Dr. Sissi C. Stove Lorentzen, Oslo University Hospital, Norway

Eldad Zacksenhaus, J Cancer Sci Ther, 3:7
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Identification of a new prognostic signatures and therapeutic targets for HER2+ and triple negative breast cancer using subtype-specific mouse models

Eldad Zacksenhaus

Toronto General Research Institute – University Health Network, Canada

HER2$^+$ and Triple-Negative Breast Cancers (TNBC) represent highly aggressive subtypes. HER2+ BC is commonly treated with chemotherapy plus anti-HER2 drugs, such as Herceptin. Drug resistance, high cost and side effects limit the use and benefits of anti-HER2-therapy worldwide. There is therefore great interest in an effective prognostic predictor and novel therapeutic targets for HER2$^+$ breast cancer. We identified MMTV-Her2/Neu mammary tumor-initiating cells (TICs) and showed that non-adherent tumorspheres can be used as surrogate for TICs (Can Res. 2007, Clin Can Res. 2009). Using improved conditions, we have further enriched Her2/Neu TICs to a frequency of 1/20-1/40. A 17-gene TIC-specific signature derived from differentially expressed genes in TICs versus non-TICs predicts survival in multiple human HER2$^+$ BC cohorts with hazard ratios of over 8, and can be used to stratify HER2$^+$ patients for anti-HER2 therapy (submitted). Using a lentiviral shRNA screen, we also identified several kinases required for Her2/Neu tumorsphere growth, for which no previous requirement for their function in HER2$^+$ BC has been described (in progress).

TNBCs often contain loss-of-function mutations/alterations in the tumor suppressors RB1, p53 and Pten. We showed that conditional deletion of Rb in mammary stem cells/bipotent progenitors led to tumors that clustered with luminal-B or TNBC. The latter contained mutations in p53. Combined deletions of Rb and p53 led exclusively to TNBC (J. Clin Invest. 2010, Cell Cycle, 2011). Similarly, inactivation of Pten in mammary epithelium induced diverse tumor types, whereas combined deletion of Pten and p53 led to TNBC-like tumors (in progress). Drug and shRNA screens have identified several agents/targets that specifically kill Rb/p53, Pten/p53 mutant TNBC as well as human TNBC lines but not immortalized mammary epithelial cells (in progress).

Biography

Dr. Eldad Zacksenhaus earned his PhD at the University of Toronto in Molecular Genetics on the cloning of UBE1, and postdoctoral training at the Hospital for Sick Children on the tumor suppressor RB. He is senior scientist at University Health Network and Associate Professor of Medicine, University of Toronto. His research is focused on tumor suppressors, in particularly Rb, breast cancer, cancer stem cells and targeted therapy for HER2 and TNBC (JCI, 2010, JCB, 2010, Cancer Res. 2010, Autophagy, 2011, PLoS One, 2011, Cell Cycle 2011). He's a co-organized of the "International RB Symposium" Nov. 17-18, 2011, Toronto, Canada.

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Susceptibility alleles of breast cancer in high, moderate and low penetrance genes in a South American population

Lilian Jara

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Breast cancer (BC) is the most common cancer among women worldwide. There are few studies of BC susceptibility alleles in South American populations. Here we describe the identification of risk variants in genes BRCA1, BRCA2 (high penetrance), CHEK2, RAD51, XRCC3 (moderate penetrance), rs2981582 and rs1219648 (low penetrance) in Chilean families at high-risk for breast/ovarian cancer (n=326). Germline BRCA1/2 point mutations were found in 7.1% of families. Families with at least three BC and/or OC cancer cases showed the highest frequency of mutation (15.9%). We identified 14 point mutations, of which 3 in BRCA1 and 3 in BRCA2 were recurrent, possibly reflecting region-specific founder effects. In CHEK2, the 1100delC mutation was not detected in the 1320 samples analyzed. The Thr241Met (XRCC3) polymorphism was associated with increased BC risk (OR=2.44, 95%CI=1.34–4.43). The RAD51 135G>C polymorphism increased BC risk among BRCA1/2-negative women with a) a family history of BC and b) age at onset <50 years (OR=2.17, 95%CI=1.11-4.29). The combined Thr/Met–E/G (RAD51D) genotype was associated with increased BC risk among the same group of women (OR=10.5 [95%CI 1.16–94.5]). Our results suggest that variability in XRCC3 and RAD51D plays a role in BC risk via a mutual interaction between the genes. The combined SNPs rs2981582 – rs1219648 (A/A–G/G) of FGFR2 genotypes were associated with increased risk for estrogen-receptor positive BC (OR=2.6, 95%CI=1.2–5.6]). Our results are consistent with a polygenic model for familial BC susceptibility.

Biography

Lilian Jara completed her PhD at 30 at a Chilean University, and postdoctoral studies at Sheffield University (UK). She is a full professor in the Human Genetics Program, University of Chile School of Medicine. She has published over 60 papers in reputed journals and serves as a WJMG editorial board member. She conducts longitudinal studies of women from Chilean families with genetic mutations related to breast cancer. She is also interested in gene-gene interactions underlining hereditary breast cancer. She collaborates with research groups studying the genetics of breast cancer from Argentina, Colombia, Spain, Canada, and the USA.

Hugo A. Barrera-Saldaña, J Cancer Sci Ther, 3:7
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Pharmacogenomics predictions for breast cancer treatments´ efficacy and toxicity

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Breast cancer is the major cause of death among Mexican women between 35 and 50 years of age. Among the most prescribed chemotherapeutic agents are Capecitabine, a precursor of 5-flourouracil, which inhibits the synthesis of thymidine and DNA replication, and tamoxifen, a chemotherapeutic adjuvant that prevents recurrence in estrogen receptor positive patients that have undergone surgery. Capecitebine needs to be efficiently eliminated by dihydropyrimidine dehydrogenase (DPD) to avoid its accumulation and adverse effects. Tamoxifen is a prodrug that needs the P450 liver enzyme isoform CYP2D6 for conversion to its active form, endoxifen. Therefore, prediction of efficacy and toxicity in breast cancer chemotherapy depends on pharmacogenomics.

PHARMAchip™ is a DNAchip developed by Progenika Biopharma, SA (Bilbao, Spain) that consists of a microscopic slide carrying hundreds of oligonucleotides to screen for mutations and single nucleotide polymorphisms in genes of phase I, phase II, transporters, receptors and other enzymes and proteins with which drugs interact after entering the patient´s body and metabolism. Among the genes present on this DNAchip genotype are those for the P450 and pyrimidine metabolism enzymes described above.

We are using the PHARMAchip to investigate its usefulness as a predictor of chemotherapy efficacy and toxicity in Mexican breast cancer patients. The first fifty genomic DNAs from these patients analyzed reveal that most (almost 85%) patients carry a genotype corresponding to a phenotype of normal to extensive metabolism of tamoxifen with no mutations in the DPD gene that would otherwise cause 5-Fluorouracil derivative accumulation.

Biography

The author completed his PhD at the age of 25 and is a distinguished alumnus from the University of Texas at Houston. His postdoctorate was at Louis Pasteur University in Strasbourg, France. He has created prestigious Molecular Biology Research and Graduate Programs and Centers in Mexico. He is the founder of Vitaxentrum, a premier Biotechnology and Genomics Consulting and Servicing Organization. He has published over 100 papers in prestigious journals and has served as reviewer of international journals and national academic committees. His team is recognized in Latin America as a pioneer and leader in molecular biology, DNA diagnostics, biotechnology, and gene therapy.

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A novel feedback loop with therapeutic implications in estrogen receptor-negative breast cancer

Ali Naderi

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Estrogen receptor-negative (ER-) breast cancer is a heterogeneous disease with limited therapeutic options. Molecular apocrine subtype constitutes 50% of ER- breast tumors and is characterized by a steroid-response signature including androgen receptor (AR) and a high rate of ErbB2 amplification. We have identified a positive feedback loop between the AR and ERK signaling pathways in molecular apocrine breast cancer. In this process, AR regulates ERK phosphorylation and kinase activity. In addition, AR inhibition results in the down-regulation of ERK target proteins including phospho-RSK1. Furthermore, AR-mediated induction of ERK requires ErbB2 and AR activity, in turn, regulates ErbB2 expression as an AR-target gene. These findings suggest that ErbB2 is an upstream connector between the AR and ERK signaling pathways. Another feature of this feedback loop is an ERK-mediated regulation of AR. In this respect, the inhibition of ERK phosphorylation reduces AR expression and CREB1-mediated transcriptional regulation of AR acts as a down-stream connector between the AR and ERK signaling pathways.

Importantly, this feedback loop has therapeutic implications in molecular apocrine breast cancer. There is an *in vitro* synergy between AR and MEK inhibitors in reducing cell viability and inducing apoptosis in molecular apocrine cells. In addition, we have demonstrated an *in vivo* synergy between AR and MEK inhibitors using a xenograft molecular apocrine model. Moreover, the combination therapy with these inhibitors can overcome trastuzumab resistance in molecular apocrine cells. Therefore, a combination therapy strategy with AR and MEK inhibitors may provide an attractive therapeutic option for ER-/AR+ subtype of breast cancer.

Biography

Ali Naderi is a clinician-scientist in oncology with a special interest in breast cancer research. He carried out his medical oncology fellowship at Mayo clinic, Minnesota and a post-doctoral research fellowship at Hutchison/MRC Research Center, University of Cambridge UK. He currently has a faculty position as a clinician-scientist at the University of Queensland, Australia. He has an American board certification in Medical Oncology and more than 25 publications in cancer research field.

M. H. Bukhari, J Cancer Sci Ther, 3:7
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Molecular classification of breast cancer and role of immunohistochemistry for detection of cell types that predicts response to chemotherapy with Santinib (P53)

M. H. Bukhari

King Edward Medical University, Lahore, Pakistan

Breast cancer is a heterogeneous group of malignant lesion resulted by abnormal gene expression within neoplastic cells. Recent advances in molecular techniques have enabled researchers to identify the gene expression, fingerprint, of individual tumors that would help predict the clinical course and select specific treatment. Molecular techniques have also been used to refine the classification of special type cancers. Four major molecular subgroups of breast cancer normal-like, luminal (ER-positive), basal-like (mostly ER-negative), or erbb2+ (mostly HER-2 amplified) have been previously defined, based on expression of 424 genes involved in cancer development. Scientists have already shown that each subgroup has a different prognosis as luminal A, luminal B, HER2 and basal-like types. Luminal A cancers are ER+ and/or PR+, HER2- and have a Ki67 labeling index <14%. Luminal B tumors are either ER+ and/or PR+ and HER2+ (the luminal-HER2 subtype) or ER+ and/or PR+ with a Ki67 labeling index >14%. HER2 tumors are ER-, PR and HER2+. As discussed below, basal-like cancers are most commonly ER-, PR-, HER2- and show expression of CK5/6 and/or EGFR.

The classification of breast cancer into molecular subgroups may be needed in order to develop the most accurate predictors of treatment response. In our experience, different sets of genes present in different molecular subgroups may determine the response to a particular regimen of chemotherapy. Luminal A type breast carcinoma shows better prognosis and best response to endocrine therapy and less response to chemotherapy. Patients with basal like are heterogeneous group of young age victims and triple negative with poor prognosis but shows good response to chemotherapy (Taxol/FAC). The cancers having extra copies of the HER2 gene and several other genes are called HER2 group. They usually have a high-grade appearance under the microscope. These cancers tend to grow more quickly and have a worse prognosis, Women with a relatively uncommon type of breast cancer are significantly more likely to face its recurrence and spread, Although they often can be treated successfully with targeted therapies such as trastuzumab (Herceptin), Santinib (P53) and lapatinib.

Biography

Dr Bukhari completed his doctorate in Surgical Pathology with the theoretical and practical combination of Histopathology, Immunohistochemistry and PCR at the King Edward Medical University in 2007. After his doctorate he attended the special course of Breast Pathology in Harvard School of Public Health in 2009. He has started work with Prof Abbas Iqbal and Eyyad H A Kamel on Chemothearpeutic effect of Sanatinib.e in triple negative patients and HER 2 Positive cases.

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Combining biologics and cytotoxics in the treatment of inoperable cholangiocarcinoma

Lars Henrik Jensen

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Cholangiocarcinomas are adenocarcinomas arising in the biliary tract epithelium either intrahepatic or extrahepatic. The disease is rare and a standard chemotherapy regime for inoperable disease has only been internationally accepted in the last few years. Based on phase III data from the ABC02 trial, gemcitabine combined with a platinum is now the treatment of choice. In other tumors from the gastrointestinal tract, the addition of newer biological compounds such as monoclonal antibodies and tyrosine kinase inhibitors has increased the efficacy of cytotoxics. Data on this approach is sparse in cholangiocarcinoma.

A comprehensive literature search identified a few studies on monotherapy with biologics. Only sorafenib, erlotinib, and lapatinib have been tested in reasonably large studies to estimate whether the drugs may be efficacious as monotherapy. The most promising drug was erlotinib with a response rate of 8%. Sorafenib has low activity with a 2% response rate while no responses were observed with lapatinib. Cetuximab and midostaurin have shown specific effect, but in a very limited number of patients.

More data is available for biologics combined with cytotoxics. Bevacizumab, cetuximab, and selumetinib have been shown to be tolerable and to have effect when combined with chemotherapy. It may not be superior to chemotherapy alone, but e.g. cetuximab have been shown to revert chemoresistance.

More phase II data preferably from randomized trials is needed in order to select the most promising combination of biologics and cytotoxics for phase III trials. International collaboration is mandatory for conducting larger trials.

Biography

Lars Henrik Jensen is a medical doctor currently working in a hospital setting serving half of the Danish population with inoperable cholangiocarcinoma. He completed his Ph.D in 2007 and has been an exchange visitor at University of Southern California. His primary areas of research are gastrointestinal cancers, clinical trials, and molecular markers.

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Stability analysis on liver cancer related miRNA in Serum

DING Xian-Feng, LI Yan and GUO Jiang-Feng
Zhejiang Sci-Tech University, P.R. China

MicroRNAs (miRNA) are non-coding, single-stranded RNAs of ~22 nucleotides and constitute a novel class of gene regulators that are found in both plants and animals. Recent evidence has shown that miRNA mutations or aberrantly expression correlate with various human cancers and indicated that miRNAs can function as tumour suppressors and oncogenes. While many studies have focused on miRNA expression in physiological and pathological processes, variables related to miRNA for new serum biomarkers have simultaneously emerged. Now miRNA has been applied to early detection of cancer and monitoring of cancer recovery by using detection of peripheral blood.

Up to present, there are no reports regarding of liver cancer specific miRNA biomarkers in serum for the great threat of liver cancer to human life. Therefore a systemic research on the characteristic of miRNA is quite necessary.

Liver cancer Huh-7 cell-line, liver tumor tissues and clinical serum samples were performed in the role of experiment material. A systemic treatment, such as different temperature(-80□, -20□, 4□, room temperature and 37□) treated for 3h, in room temperature treated for 0, 1, 3, 6, 12, 24 hours, RNase A treated for 0, 3, 6, 12 hours incubation in 37□, DNase I treated for 0, 3, 6, 12 hours incubation in 37□, different free-thaw cycles (0, 2, 5, 7, 10 cycles) treated, different pH value (control, pH=1, 6, 9, 13) of solution treated for 3h incubation in 37□ were performed before Quantitative Reverse Transcription Polymerase Chain Reaction (qRT-PCR) analysis with 40 cycles. Furthermore, liver cancer related miRNAs were detected in each reaction. We used 18S rRNA as control gene. All the results indicated that 18S rRNA was fragile for decreased relative expression sharply. MiRNAs could be resistant to harsh conditions simultaneously. The p-value indicated the repeatability of the data by using the Student's t-test. $P < 0.05$ was considered to be significant.

Meanwhile, we evaluated the Pearson's correlation coefficient of liver cancer related miRNAs expression of 22 healthy human subjects by qRT-PCR. Expression levels of serum miRNAs were reproducible and consistent among 22 healthy human subjects for the R value was access to 1, p-value □0.05. The result was considered to be significant. Pearson correlation scatter plot of the relative serum miRNAs expression between male and female of R^2 was 0.0953. Results suggested that miRNA expression is not correlated between genders.

Taken together, these results implied that liver cancer related miRNA (miRNA-21, -25, -29c, -93, -198, -221, -222) expression levels in serum were quite stable, also present and detectable, reproducibly consistent among individuals of the same species in serum. They will be potential for serum liver cancer biomarkers in future.

On the other side, it is difficult to obtain abounding and high-quality of RNA in serum. Our tests have shown that pre-heating procedure is a robust serum RNA extraction method and efficient for RNA isolation. This method is also essential for further serum source microRNA study. In fact, after qRT-PCR, the C_T (threshold cycle) value decreased at least 5. In conclusion, based on our study, pre-heating provided an optimized protocol for serum RNA isolation.

Biography

Xianfeng Ding is an associate professor of Biology, and is Director of the Biological Science Experiment center at the Zhejiang Sci-Tech University,Xianfeng Ding holds a BS degree in chemistry from Shanxi Normal University and a MD degree in Chemistry from Jiangnan University ; Pursuing research in the interdisciplinary areas of chemistry and biology, she did some research biochip technology for novel methods in emerging life and medical science fields by biochip technology at Dr. Gao' lab at University of Houston, seeking for a potential molecular biomarker for liver injury, correlation and quantitation of microRNA aberrant expression in tissues and sera from different tumors, research has been continuously funded by the National High-Tech Research & Development Program of China (Grant No. 2007AA02Z165) and Zhejiang Nature Science Foundation of China((Grant No.Y2100681), She has published more than 20 papers in reputed journals, has more than six issued and pending patents and is general manager of biotechnology companies with success in product commercialization.

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The status of anti-metastatic gene therapy in patients with advanced epithelial ovarian cancer

Samir A. Farghaly
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Ovarian cancer is the foremost cause of death from gynecological cancer in the developed world. In the USA 27,000 new cases of ovarian cancer, and 14,000 deaths are reported each year. About 80% of patients with ovarian cancer present with metastatic disease. The overall 5-year survival rate for women with cancer is 30%. The epithelial cells of the ovary constitute 1% of the total ovarian mass but constitute 90% of the ovarian neoplasms. Epithelial ovarian cancer (EOC) spreads initially by direct extensions into adjacent organs, especially the fallopian tubes, uterus, and contralteral adenexa and occasionally the rectum, bladder, and pelvic side wall . After direct extension, epithelial ovarian cancer frequently disseminates via transcoelmic route, with 70% of patients having peritoneal metastases at staging laparotomy. The correlation between molecular profiles and metastatic spread varies depending on tumor type and metastatic site and is combination of 2 models. First, tumors are genetically heterogeneous and that metastases arise from colones with a genetically acquired metastatic phenotype, and that the clonoal genotype determines the final site of metastases. The second model is that metastatic cells are not a genetically primary tumor, instead they arise as stochastic event, with a low but finite probability from tumor cell clones distinct from the primary tumor. Several cofactors , such as MMP-2/-9 inhibitor, TNF, lypmphotoxin a, Fas Ligand Fas L, APO3L, TRAIL, interleukin -8, and P38 MAPK regulating ovarian cancer cells attachment to omentum and /or peritoneum have been identified, and would have noticeable clinical inhibition of the metastatic process, by enabling the identification of cellular or molecular targets that therapeutically viable. That would be able to block the steps necessary for ovarian cancer metastasis within the peritoneal cavity.

Biography

Samir A. Farghaly is a physician / Scientist- faculty member at the Medical College of Cornell University, and the New York Presbyterian Hospital/ Cornell University Medical Center, New York, NY – USA. He received his M.D degree from University College, London University and his PhD degree in molecular biology from London University. He was affiliated with Columbia University College of Physicians and surgeons/ Columbia University medical center, New York, NY. He received several clinical and research awards. He has been an invited speaker in several national and international conferences on Women's health, Molecular genetic of female cancers, Gynecological cancer and Oncology. He is a member of several national and international societies, organizations, foundations of Women health and Cancer. He is an editor, member of editorial boards, editorial advisory boards and reviewers of several medical journals of Cancer Science & Therapy, Gynecology, Gynecological Cancer, Ovary Research, Genomics, Clinical & Experimental Obstetrics and Gynecology, and Oncology. He has published 78 articles in reputed peer review journals. He is an editor of a book on ovarian cancer (To be published in 2011).

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Detection and quantification of circulating melanoma cells in patients with cutaneous malignant melanoma

M Ziman, M. Millward, R. Pearce, M. Lee, P. Kumarasinghe and A. Ireland
Edith Cowan University, Western Australia

Our research is aimed at detection, characterisation and quantification of circulating melanoma cells in patients with Cutaneous Malignant Melanoma. This research will assist with development of a prognostic blood test for the detection of melanoma micrometastases in patients before and after surgery and during therapy to assess efficacy. To date we have used qRT-PCR to assess circulating melanoma cells in 300 melanoma patients and 100 healthy volunteers. The frequency and level of expression of markers was correlated to Breslow tumour thickness and tumour progression and results were statistically analysed. Control blood samples spiked with cells from metastatic melanoma cell lines were used as positive controls. Antibodies to melanoma cell markers have also been used in flow cytometry and immunomagnetic bead capture experiments to isolate circulating cells from patient blood samples. Our results clearly demonstrate the presence of circulating melanoma cells in 79% of patients with stage III and IV disease whilst these markers were observed in only 20-30% of early stage patients. Assay sensitivity tests showed that markers can be detected from as few as 5 cells per blood sample. Surprisingly, melanoma cells are found in peripheral blood of patients with early stage tumours and in patients from whom tumours were removed several years previously. Using immunomagnetic bead capture we have quantified the circulating cells in patient blood and found that cell number correlates with disease stage particularly when specific cell surface markers are used to isolate cells. Further research will clarify the molecular signature of metastatic circulating melanoma cells.

Biography

Mel Ziman completed her Ph.D at the University of Cape Town and postdoctoral studies from the University of Western Australia School of Medicine. She is the director of ECU Melanoma Research Foundation, a leading melanoma research group in Western Australia. She has published more 70 papers in reputed journals and serves as a reviewer for several journals and grant review panels worldwide.

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A high prevalence of metabolic syndrome in a group of colorectal patients pre surgery– A pilot study

Sissi C. Stove Lorentzen, Ingunn Bergstad, S.Gharagozlian
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Introduction: Colorectal cancer (CRC) is considered a cancer of high income countries and several environmental factors including body fatness and abdominal fat are associated with CRC. Nutritional status of the patients will have an impact on the postoperative recovery period and long term survival. A pilot study was designed to evaluate the appropriateness of commonly used physical and nutritional screening tools on the colorectal patient population at the time of the pre-surgery assessments.

Methods: Over a 6 month recruitment period, 29 patients (median age 65 (62;72) were included into the study. Baseline nutritional status was determined for all patients before intervention by using Subjective Global Assessment (SGA), Body mass index and waist circumference. Metabolic syndrome was determined using the International Diabetes Foundation criteria. Selected biochemical markers and physical tests were also included.

Results: Twenty-two patients (76 %) were classified as well nourished and seven patients (24 %) as moderately malnourished. Sixteen patients (55 %) had metabolic syndrome. Among the physical tests, the handgrip test was significantly associated with SGA ($p = 0.02$).

Conclusion: The results from the pilot confirm results from other studies which show that CRC patients may suffer from overnutrition as well as undernutrition. SGA should not be the only screening in assessing CRC patients prior to surgery. The high number of patients with metabolic syndrome must be confirmed, however, this knowledge suggests a necessity for nutritional counselling in this patient group post surgery, which is in accordance with the World Cancer Research Fund recommendations of 2007.

Biography

Sissi Stove Lorentzen has completed her MS /RD at the age of 48 from the University of Oslo in 2009. She is currently working at Oslo University Hospital as a clinical nutritionist/dietitian in the field of surgery and cancer. Her master thesis was a pilot study which investigated the effect of perioperative nutrition given to a group of colorectal cancer patients.

Sedegheh Gharagozlian has completed her Ph.D at the age of 50 years from Oslo University. She has published 3 papers in reputed journals and has been served as a journal referee for 2 journals. She has worked at Oslo University Hospital as a clinical nutritionist/dietitian in the field of gastrointestinal surgery and intensive nutrition, since 1998.She is a member of the Norwegian scientific committee for food safety. She is also a member of Advisory group in Oslo university hospital to develop a common guideline for ICU.