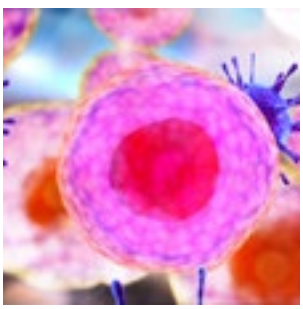
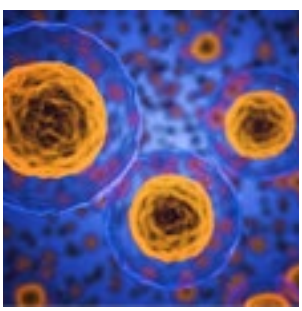
Poster
June 13, 2022

Cancer Summit 2022

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Effect of Co-culturing both placenta-derived mesenchymal stem cells and HepG2 cells in cancer cell (HepG2) migration, damage through apoptosis, cell cycle arrest

F A Dain Md Opo[1], Mohammed Moulay[1], Ghadeer Alrefaei[1,2], Nouf Hamid Alsubhi[1] and **Saleh Alkarim[1]**
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Human placental-derived mesenchymal stem cells (hPMSCs) are a promising candidate to inhibit the proliferation of hepatocellular carcinoma (HCC) cell lines such as HepG2. However, the effects of hPMSCs and their conditioned media on HepG2 are still elusive. Therefore, this study aimed to investigate the effects of hPMSCs and their conditioned media on HepG2 and elucidate the underlying mechanism of action. The percentage of cell death (early apoptosis, late apoptosis) was observed by fluorescence-activated cell sorting and MTT assay. The DIO and DID color were used to detect interaction and cell death of both cells through cell fusion. Co-treatment of HepG2 cells with hPMSCs or hPMSCs-conditioned medium (hPMSCs-CM) inhibited HepG2 proliferation and induced their apoptosis. Morphological changes were also observed in the case of 30%, 50%, and 70% co-culture of both cells together *in vitro*. Treatment with hPMSCs or hPMSCs-CM induced HepG2 cell death through apoptosis as detected by flow cytometry, caspase 9 immunofluorescence, qPCR (detection of Bax, Bcl-2, and B catenin genes), by western blot, immunophenotyping (detection of caspase 9, caspase 3 protein). The hPMSCs and hPMSCs-CM could induce HepG2 cell cycle arrest. HepG2 cell growth was arrested in the G0/G1 phase following treatment with hPMSCs or hPMSCs-CM. These treatments also inhibited the migration of HepG2 cells with maximum effect when using the highest ratio/concentration of hPMSCs (70%) and hPMSCs-CM (90%). Our results suggested that hPMSCs and hPMSCs-CM will be promising candidates to treat liver cancer..

Recent Publications

1. F A Dain Md Opo, et.al, (2022): Comprehensive Studies of Different Cancer Diseases among Less-Developed Countries. Healthcare (Basel) ;10(3):424

2. F A Dain Md Opo, et.al, (2021): Cytotoxicity Study of Cadmium-Selenium Quantum Dots (Cdse QDs) for Destroying the Human HepG2 Liver Cancer Cell. J Biomed Nanotechnol;17(11):2153-2164

3. F A Dain Md Opo, et.al, (2021): Structure based pharmacophore modeling, virtual screening, molecular docking and ADMET approaches for identification of natural anti-cancer agents targeting XIAP protein. Sci Rep ;11(1):4049..

Biography

F A Dain Md Opo is a Ph.D. Graduate at King Abdul-Aziz University in 2019. He is a member of the global collaborative research team based on the Novel Global Community Educational Foundation (NGCEF), Australia. Currently working in the cancer stem cell unit, King Fahd Medical Research Center from 2019. His research field includes stem cell biology, cancer biology, molecular biology techniques, animal handling (*in-vivo*), type 2 diabetes, cancer informatics, and Nano product modification for efficient drug delivery against cancer. His publication was included in several renowned journals (more than eight) from the beginning of his research career in 2014. He currently works on two projects, Stem cells effect against several cancers and the discovery of new natural compounds through EGFR targeting. His research interests are Stem cells, Cancer informatics, Cancer biology, Nano-products, and Cancer stem cell.

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The intermediate filament: BFSP1 is expressed in various cancer cell lines and solid tumours

Balazs Veres, Gyongyi Nagyne Kiss, Bence Kiss, Viola Bagone Vantus, Zita Bognar, Ferenc Gallyas and **Antal Tapodi**
University of Pecs Medical School, Hungary

BFSP1 (beaded filament structural protein one, or Filensin) is an eye lens-specific, cytoskeletal protein, which forms intermediate filaments (IFs) with its assembly partner (BFSP2) in the fiber cells of the eye lens. Previously, we proved that Filensin is a substrate for Caspases, which exposed an internal N-myristoylation site of the Tail-fragment of BFSP1. Our group identified D433 and D549 caspase cleavage sites releasing the main 53kDa N-terminus domain and two tail fragments: G434-D549 and G550-S665. Filensin is processed by caspases under physiological conditions as well during the development of the eye lens. Here we demonstrate that splice variants of BFSP1 are also expressed in various cancer cell lines which were proved by Western blotting, Q-PCR, and Mass spectrometry. BFSP1 and proteolytic fragments showed remarkable membrane binding, which was confirmed *in vitro* in cancer cell lines and ex vivo in human breast carcinomas. According to the literature, BFSP1 has been known as an intermediate filament expressed exclusively in eye lenses so far. The appearance of BFSP1 in cancer cells seems very unique and it indicates a new exciting approach in the field of tumor biology. To establish the possible role of BFSP1 expressed in tumor cells might have extraordinary significance in the tumor diagnosis and it could provide a new possible target in tumor therapy.

Recent Publications

1. Balazs Veres, et.al, (2021). Cyclophilin D-dependent mitochondrial permeability transition amplifies inflammatory reprogramming in endotoxemia. FEBS Open Bio;11(3):684-704.

2. Balazs Veres, et.al, (2015). Anti-inflammatory effects of a triple-bond resveratrol analog: structure and function relationship. Eur J Pharmacol; 748:61-7.

3. Balazs Veres, et.al, (2003). Decrease of the inflammatory response and induction of the Akt/protein kinase B pathway by poly-(ADP-ribose) polymerase 1 inhibitor in endotoxin-induced septic shock. Biochem Pharmacol: 65(8):1373-82.

Biography

Balazs Veres is working at the Department of Biochemistry and Medical Chemistry, University of Pécs Medical School, Hungary, and is well experienced in his field and has published many articles.

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Design and construction of recombinant neoepitope tumour antigen in order to raise polyclonal antibodies against BFSP1

Antal Tapodi, Bence Kiss, Gyongyi Nagyne Kiss, Viola Bagone Vantus, Balazs Veres and **Ferenc Gallyas**
University of Pecs Medical School, Hungary

BFSP1 (beaded filament structural protein one, or *Filensin*) is an eye lens-specific cytoskeletal protein, that forms intermediate filaments (IFs) with its assembly partner (BFSP2) in the fiber cells of the eye lens. Surprisingly, BFSP1 was identified in cultured human tumor cells "*in vitro"* as well. Previously, we proved that BFSP1 is expressed in cultured cell lines, as well as "*ex vivo*" human solid tumors by Western Blotting and MS. Furthermore, we quantitatively determined the relative expression of BFSP1 splice variants by qPCR from different cell lines. In order to provide reliable positive control, we cloned and expressed recombinant BFSP1 splice variants in a prokaryotic expression system. Since there is no sufficient antibody against BFSP1, we produced polyclonal antibodies with a BFSP1 antigen presenting self-assembling protein nanoparticle (SAPN) using rabbits. The novel antibodies were tested via Western blotting and will be essential for further research. According to the literature, BFSP1 has been known as a cytoskeletal protein expressing particularly in eye lenses so far. The presence of BFSP1 in cancer cells seems unlikely and it indicates a new exciting approach in the field of tumor biology. To establish the possible role of a new cytoskeletal protein as a tumor marker might have extraordinary significance in cancer diagnosis..

Recent Publications

1. Antal Tapodi, et.al, (2022). Involvement of Mitochondrial Mechanisms and Cyclooxygenase-2 Activation in the Effect of Desethylamiodarone on 4T1 Triple-Negative Breast Cancer Line. International Journal of Molecular Sciences. 23. 1544. .

2. Antal Tapodi, et.al, (2020). Amiodarone's major metabolite, desethylamiodarone inhibits proliferation of B16-F10 melanoma cells and limits lung metastasis formation in an *in vivo* experimental model. PloS one. 15.

3. Antal Tapodi, et.al, (2019). BFSP1 C-terminal domains released by post-translational processing events can alter significantly the calcium regulation of AQP0 water permeability. Experimental Eye Research. 185.

4. Antal Tapodi, et.al, (2018). PARP inhibition induces Akt-mediated cytoprotective effects through the formation of a mitochondria-targeted phospho-ATM-NEMO-Akt-mTOR signalosome. Biochemical Pharmacology. 162.

Biography

Antal Tapodi is working at the Department of Biochemistry and Medical Chemistry, University of Pécs Medical School, Hungary, and is well experienced in his field and has published many articles.

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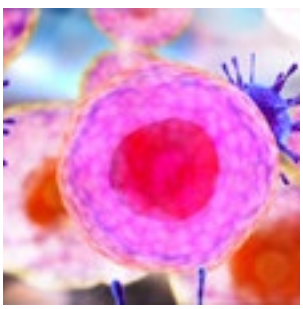


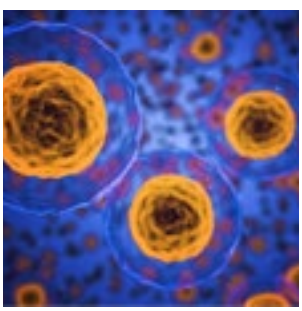


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Decreased cell proliferation with 2, 3- Dichloro-5, 8-Dimethoxy-1, 4-Naphthoquinone and 4-Hydroxy Tamoxifen in triple negative breast cancer cell lines

Robert L Copeland
Howard University, USA

Triple negative breast cancer (TNBC) is a subtype of breast cancer (BC) that makes up 10-15% of BC diagnosis. This form of the disease disproportionately affects African American (AA) women as compared to women of other ethnicities. TNBC is classified by lack of expression of three receptors: As specific targets are not present, surgery, radiation, and chemotherapy have been the mainstay of therapy for TNBC. A novel compound, 2, 3-dichloro-5, 8-dimethoxy-1, 4-naphthoquinone (Z285) belongs to the class of naphthoquinones that demonstrate activities as anticancer compounds. These compounds have been shown to cause an increase in reactive oxygen species (ROS) production as well as effects signaling pathways associated with epidermal growth factor receptor (EGFR). Moreover, it has been reported that treatment with tamoxifen can cause cell death. This is based on the accumulation of the drug and its active metabolites within cells that may cause an increase in the ROS production ultimately leading to cellular death, even in ERα negative cell lines.

In the current study, three triple negative cell lines, HCC1806, MDA MB 231, and HS578T were used. These cells were treated with Z285 and 4-hydroxy tamoxifen (4OH-Tam) for 24 or 72hrs with 1, 2, 4, 8, 16µM, and 3, 6, 12, 24, 48µM respectively. Synergestic activity was demonstrated using SynergyFinder. In other experiments, cells were treated with the IC50 of Z285 which was established in prior experiments. mRNA and protein were isolated between 6 and 24hrs. Results showed alterations of several mRNAs in response to these treatments. In addition, there were modifications to Nrf2 protein concentration, a transcription factor associated with oxidative stress. Therefore, these results indicate that combination treatment of Z285 and 4OH-Tam induces cell death at lower concentrations. Thus, this novel compound which causes an increase in ROS in these TNBC cells may render them more susceptible to tamoxifen therapy.

Recent Publications

1. Robert L Copeland, et.al, (2021): A panel of miRNAs as prognostic markers for African-American patients with triple negative breast cancer. BMC Cancer. 21(1):861.

2. Robert L Copeland, et.al, (2021): New targets in triple-negative breast cancer. Nature Reviews Cancer. 21(12).

3. Robert L Copeland, et.al, (2021): Sodium Butyrate Protects Against Ethanol-Induced Toxicity in SH-SY5Y Cell Line. Neurotoxicity Research. 39(2).

Biography

Robert Copeland has completed his PhD from Howard University, USA. He is the Associate Professor and Chair of the Dept of Pharmacology, Coll of Medicine. He has over 80 publications that have been cited over 700 times, and his publication H-index is 13 and has been serving as an editorial board member of reputed Journals. His research efforts are in directions that will delineate molecular differences in breast cancers amongst the two ethnic groups (African Americans and European Americans) which will help identify ethnic-specific markers for breast cancer progression. The major focus is on the development of novel pharmacological approaches for the prevention and treatment of breast cancers thus developing a more tailored treatment approach leading to better management of breast cancer.

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Can performance of an ai algorithm be improved on a specific ethnicity after focused training?

Mohamad Hajaj
Jarvis Breast Screening Centre, UK

Clinical Relevance Statement: Training machine learning algorithms requires varied datasets to prevent ethnic bias. Fine-tuning of algorithms with small datasets via transfer learning offers a viable solution to algorithmic bias.

Purpose: Screening mammography remains the only effective screening method that significantly improves patient mortality. The accuracy of mammograms can vary between experts, resulting in inconsistent interpretations. Multiple artificial intelligence (AI) algorithms are being developed to assist in cancer detection, but they may have training bias due to limited ethnic diversity. We hypothesize that AI algorithms can be optimized for a specific patient population by using various fine-tuning strategies that utilize transfer learning.

Materials and Methods: A test set of 100 random 2D FFDM mammograms of Arab women was obtained between July 2019 and December 2020. This was used to test a baseline novel AI algorithm which was trained on a large multi-ethnicity dataset, resulting in a Global Prediction Score of Malignancy for each mammogram. Then, the layers of the deep learning AI algorithm were fine-tuned to three different extents: all Layers, some Layers and few Layers using additional 500 random mammograms of Arab women obtained in a similar manner and period. The three fine-tuned AI algorithms were then each re-tested on the test set, and their respective Global prediction Scores were compared to that of the baseline AI algorithm. Results were evaluated and compared using sign test, Wilcoxon test, and percent change error metrics as well as by visualizing absolute error across cases.

Results: Absolute error for all test cases were computed. The three fine-tuned algorithms: all layers, some layers, and few layers resulted in better classifying the cases for 40%, 31%, and 32% of the times, respectively. The reduction in error rates is 5.2%, 14.6%, and 11.2% respectively.

Conclusion: Fine-tuning of the AI algorithm using transfer learning and a small sample of Arabic patients showed a trend towards improved case classification and a reduction in error when tested on other Arabic patients. The benefit of fine-tuning the layers of deep learning algorithm using transfer learning optimizes the algorithm for better performance for a specific patient population. This concept should be explored further to develop ethnically unbiased AI algorithm in medical imaging applications.

Recent Publications

1. Ginawi, A., Hajaj, M., Pascaline, S., Woodland, K., & Dakka, M. (2017). Contrast Enhanced Spectral Mammography – A UK centre experience. European Journal of Surgical Oncology (EJSO), 43(5), S19–S20.

2. Pascaline, S., Patel, S., Horvath, K., & Hajaj, M. (2016), Preoperative role of Contrast Enhanced Mammography (CESM) in breast cancer, European Journal of Surgical Oncology (EJSO), 43(5), S19–S20.

Biography

Mohamad Hajaj is senior consultant radiologist who has joined Hamad Medical Corporation in December 2017. Dr. Hajaj has been working in the UK for the last 12 years in different roles including consultant radiologist, Clinical Director of Radiology Department, Director of the Breast Screening Services, and Clinical Lead for Governance. These positions were in highly reputable hospitals such as the University Hospitals of Leicester, Kettering General Hospital NHS, North Nottinghamshire Hospitals, North Staffordshire University Hospitals and Bart's London.

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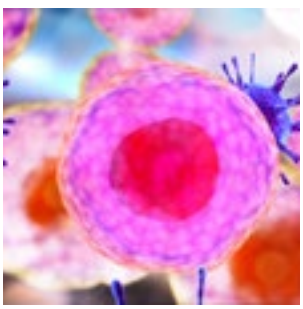
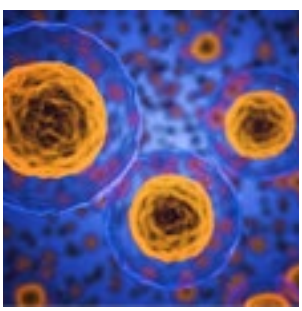
Accepted Abstracts

Cancer Summit 2022

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Combinatorial CRISPR profiling of driver gene permutations that underlie breast cancer

Scott Powers
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Most common human adult cancers involve the alteration of multiple driver genes. Certain features of how driver genes collaborate to promote cancer are known, including their relative timing during cancer progression and their functions in different oncogenic signaling pathways. However, the degree to which cancer results from genetic interactions (epistasis) between driver genes as opposed to the sum of individual driver gene effects is largely unknown. Greater knowledge of epistatic interactions among driver gene alterations is necessary to accurately predict which phenotypes and therapeutic vulnerabilities are to be expected based on a patient's cancer genome. However, uncovering these epistatic interactions and understanding their contribution to cancer progression has not been possible until the development of combinatorial CRISPR profiling that integrates CRISPR phenotypic screening with single-cell transcriptome readouts. We used this methodology to systematically analyze how combinations of inactivated tumor suppressor genes (TSG) changed the growth properties and gene expression profiles of human mammary epithelial cells, with the goal of identifying general mechanisms of driver gene cooperation. We prepared several derivatives of the commonly used nontumorigenic breast epithelial model, MCF10A. Derivatives of MCF10A were infected with a combinatorial CRISPR library targeting 52 TSGs and cultured *in vivo* by injection into murine mammary fat pads. These cells with diverse genotypes then competed with one another for 6 to 8 weeks, allowing for the identification of epistatic interactions, which are the pairwise perturbations that result in faster than expected tumor cell growth. Surprisingly, the epistatic interaction networks were comprised of numerous cliques—sets of three or four genes such that each TSG within the clique showed oncogenic cooperation with all other genes in the clique. Single-cell transcriptomic profiling of CRISPR double knockouts revealed that cooperating TSGs that synergized in promoting tumorigenesis showed transcriptional epistasis, whereas noncooperating TSGs did not. These epistatic transcriptional changes, both buffering and synergistic, affected the expression of oncogenic mediators and therapeutic targets, including CDK4, SRPK1, and DNMT1. Importantly, the epistatic expression alterations caused by dual inactivation of TSGs in this system, such as PTEN and TP53, were also observed in patient tumors, establishing the relevance of these findings to human breast cancer. Overall, our study indicates that transcriptional epistasis is a central aspect of multigenic breast cancer progression and provides a roadmap for moving beyond the discovery and development of therapeutic strategies based on single.

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Breast specific ELF5 clock for the risk assessment to Breast Cancer

Masaru Miyano, Rosalyn W Sayaman and Mark A LaBarge
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Breast Cancer (BC) is the most common cancer among women in the U.S. A robust BC prevention strategy requires risk assessment biomarkers for early detection. More than 75% of BC diagnoses occur in women over 50 years of age, suggesting that aging is the greatest risk factor for BC. However, biomarkers that are currently used are unable to precisely measure an older individual's physiological or functional age. Thus, we are motivated to identify a biomarker to predict the risk of BC and simultaneously develop a prevention tool. During aging in the human mammary gland, luminal epithelial cells lose lineage fidelity as evidenced by decreased expression of luminal lineage-specific genes and increased expression of markers unique to myoepithelial cells. One prominent feature of the loss of luminal lineage fidelity with age is the downregulation of luminal-specific ELF5 gene expression with increased DNA methylation on its promoter. ELF5 is central to the maintenance of healthy luminal epithelial cells and shows age-dependent changes that parallel changes seen in age-related BC. We have reported that both ELF5 expression and methylation can be used to build biological clocks to estimate the chronological ages of mammary epithelia. ELF5-clock-based estimates of biological age in luminal epithelia from average-risk women were within three years of chronological age. Using the same clock, the biological ages of breast epithelia from BRCA1 or BRCA2 mutation carriers, who have a high risk of developing BC, are accelerated by two decades relative to chronological age. Our finding indicates that the changes in ELF5 expression or ELF5-proximal DNA methylation in luminal epithelia are emergent properties of at-risk breast tissue and constitute a breast-specific biological clock.

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CRISPR screening of CAR T Cells and cancer stem cells reveals critical dependencies for cell-based therapies

Dongrui Wang
City of Hope Medical Center, USA

Glioblastoma (GBM) contains self renewing GBM stem cells (GSCs) potentially amenable to immunologic targeting, but chimeric antigen receptor (CAR) T cell therapy has demonstrated limited clinical responses in GBM. Here, we interrogated molecular determinants of CAR-mediated GBM killing through whole-genome CRISPR screens in both CAR T cells and patient-derived GSCs. Screening of CAR T cells identified dependencies for effector functions, including Transducin-like enhancer protein 4 (TLE4) and IKAROS Family Zinc Finger 2 (IKZF2). Targeted knockout of these genes enhanced CAR antitumor efficacy. Bulk and single cell-RNA sequencing of edited CAR T cells revealed transcriptional profiles of superior effector function and inhibited exhaustion responses. Reciprocal screening of GSCs identified genes essential for susceptibility to CAR-mediated killing, including RELA and NPLOC4, the knockout of which altered tumor-immune signaling and increased responsiveness of CAR therapy. Overall, CRISPR screening of CAR T cells and GSCs discovered avenues for enhancing CAR therapeutic efficacy against GBM, with the potential to be extended to other solid tumors.

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Targeting mutant p53 degradation for cancer therapy

Shengliang Zhang
Brown University, USA

The tumor suppressor TP53 is mutated in over 50% of human tumors. Most of the p53 mutations are missense mutations. Mutant p53 protein not only loses the wild-type functions but also gains new oncogenic properties that contribute to tumorigenesis, tumor progression, and drug resistance. Targeting mutant p53 by either restoring the p53 pathway and/or depleting its gain-of-function is a promising strategy for cancer therapy. We conducted a high-throughput screening using multiple chemical libraries and identified a small molecule NSC59984 as a promising lead compound with the dual capability to restore p53 pathway signaling and destabilize mutant p53 in cancer cells. NSC59984 induces mutant p53 degradation via the MDM2-mediated Ubiquitin-proteasome pathway. A reactive oxygen species (ROS)-ERK2 axis is required for NSC59984 to induce the MDM2-dependent mutant p53 degradation. These discoveries propose that an inducible ROS-ERK2-MDM2 axis exposes a vulnerability in mutant p53 stabilization and can be exploited by small-molecule compounds to induce mutant p53 degradation for cancer therapy. Tumor suppressor p73, a member p53 family can transcriptionally activate many p53-targets, therefore, p73 appears to be a promising target to reinforce p53 pathway signaling bypassing restoration of wild function to mutant p53. The mutant p53 degradation releases p73 from the mutant p53 inhibitory complex. Small molecule NSC59984 restores p53 pathway signaling via enhancing p73 transcriptional activity. Our xenograft tumor models demonstrate that NSC59984 suppresses tumor growth via p73, and the high cellular ROS increases the efficacy of NSC59984 antitumor effects. Overall, our studies provide a therapeutic strategy for activating and releasing p73 from a mutant p53 inhibitory complex by induction of mutant p53 degradation in cancer cells.

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Indium oxide nanoparticles (In2O3 NPs) - 2

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Indium oxide nanoparticles (In2O3 NPs) is being studied for a variety of applications including gas-sensing, environmental remediation, and biomedicine. It is aimed to examine the effect of silver (Ag) doping on the photocatalytic and anticancer activity of In2O3 NPs. The Ag-doped (2%, 4%, and 6wt%) In2O3 NPs were synthesized by the photo-deposition method. Prepared samples were characterized via X-ray diffraction (XRD), transmission electron microscopy (TEM), scanning electron microscopy (SEM), X-ray photoelectron spectroscopy (XPS), Fourier transform infrared (FTIR), UV-vis spectrometer, and photoluminescence (PL). XRD data showed that Ag-doping increases the crystallinity of In2O3 NPs. SEM and TEM images indicated that In2O3 NPs have a spherical shape with smooth surfaces, and Ag-doping increases the size without affecting the particle's morphology. XPS spectra showed the oxidation state and the presence of Ag in In2O3 NPs. Bandgap energy of In2O3 NPs decreases with increasing the concentration of Ag (3.41 eV-3.12 eV). The peak intensity of PL spectra of In2O3 NPs also reduces with the increment of Ag ions suggesting the hindrance of the recombination rate of e-/h+. The photocatalytic activity was measured by the degradation of RhB dye under UV irradiation. The degradation efficiency of Ag-doped (6%) In2O3 NPs was up to 92%. Biochemical data also indicated that Ag-doping enhances the anticancer performance of In2O3 NPs against human lung cancer cells (A549). Overall, this study demonstrated that Ag-doping enhances the photocatalytic activity and anticancer efficacy of In2O3 NPs. This study warrants further investigation of environmental and biomedical applications of Ag-In2O3 NPs.

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The apoptotic effect of Ferulic acid-synthesized gold nanoparticles against human epidermoid carcinoma (A431) cells via activation of caspase-3 pathway

Lonchin Suguna, Indra Rajendranan, Thangavel Ponrasu and **Rama Rajaram**
Central Leather Research India, India

In the present study, ferulic acid, a polyphenol, was employed to prepare stable gold nanoparticles. It acts as a reducing agent when mixed with hydrogen tetrachloroaurate (III) hydrate at ambient temperature. Subsequently, fa also turned into a stabilizing agent and yielded spherical gold nanoparticles (fa-AuNPs). The synthesized gold nanoparticles (fa-AuNPs) were thoroughly characterized using UV/Visible spectroscopy, Scanning electron microscopy, High-resolution transmission electron microscopy, Dynamic light scattering, and Fourier-transform infrared spectroscopy studies. Then, the synthesized fa-AuNPs were tested in human skin cancer cells (A431) and normal kertotinocytes (HaCaT cells). The fa-AuNPs produced cytotoxicity in A431 cells in a dose and time-dependent manner. The angiogenetic efficacy of the fa-AuNPs was substantiated by the results of the CAM assay. The programmed cell death occurred via apoptosis as indicated by the sub-G1 population. Increased levels of reactive oxygen species and caspase-3 activity resulted in reduced mitochondrial membrane potential. Hence, this study corroborated that fa-AuNPs successfully stimulated autophagy in A431 cells through mitochondria-based pathways and thus may be considered a potential agent to treat skin cancer.

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GC-MS analysis of marine invertebrate from sea urchin (*Diadema savignyi*) and identification of potential anti-cancer activity against colorectal cancer

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Colorectal cancer is the second most common chronic disease in the world that affecting almost 1.9 million people in 2020. Till now, no specific drug candidates have been developed or yet available that can treat this cancer. Therefore, it is an urgent need to discover a novel anticancer drug against the diseases from the marine invertebrate. However, Importin-11 is a protein that is responsible for transporting β-catenin to the cell nucleus and acts as a cell proliferation of colorectal cancers. The blocking of Importin-11 can block the β-catenin from entering the nucleus. It may act as a precursor to inhibit the growth of colorectal cancer formed by APC mutant. Therefore, the study aims to identify potential natural anticancer agents that can inhibit the activity of Impotrin-11, subsequently blocking the progression of colorectal cancer. Initially, a total of 15 compounds from the Sea Urchin invertebrates were identified through the gas chromatography-mass spectrometry (GC-MS) analytical method. Consequently, the compounds were screened through molecular docking, absorption, distribution, metabolism, excretion (ADME), toxicity (T), and molecular dynamics (MD) simulation approach. The molecular docking method initially identified four molecules having PubChem CID: 304, CID: 6432458, CID: 605775 and, CID: 11955 with a good binding affinity. All the selected compounds exhibit good pharmacokinetics and toxicity properties. Finally, the four compounds were further evaluated based on the MD simulation methods that confirmed the binding stability of the compounds to the targeted protein. The computational approaches identified the best four compounds CID: 304, CID: 6432458, CID: 605775, and, CID: 11955 that can be developed as a treatment option that has a better binding affinity to the target protein. To sum up, marine animal sea urchin showed better anticancer activity against an importein-11 protein that can be further developed as an anti-CRC drug. Although, experimental validation is suggested for further evaluation of the work.

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Vincosamide suppresses malignant behaviors of hepatoma cells by activating caspase-3 activity and blocking the PI3K/AKT signaling pathway

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Background: *Vincosamide* (Vinco) was first identified in the methanolic extract of the leaves of *Psychotria leiocarpa* and Vinco has important anti-inflammatory effects and activity against cholinesterase, Vinco also has a trait to ant-tumor. However, whether Vinco can inhibit the malignant behaviors of hepatocellular carcinoma(HCC) cells is still unclear. In the present study, we explored the role of Vinco in suppressing the malignant behaviors of HCC cells.

Methods: Microculture Tetrazolium Assay (MTT), trypan blue exclusion assay, the Cell Counting Kit (CCK)-8 and flow cytometric analysis were applied to detect the proliferation and death of HCC cells; electron microscopy was performed to observe the change in cellular mitochondrial morphology; scratch repair and Transwell assays were used to analyze the migration and invasion of HCC cells; expression and localization of proteins were detected by laser confocal microscopy and Western blotting; the growth of the cancer cells *in vivo* was assessed in a mouse tumorous model.

Results: At a dose of 10-80 μ g/ml, Vinco inhibited the proliferation, migration, and invasion and promoted apoptosis of HCC cells in a dose-independent manner, but had a low cytotoxicity effect on normal liver cells. Silenced expression of alpha-fetoprotein (AFP) could promote Vinco inhibits the proliferation of HCC cells. Additionally, 80 μg/ml of Vinco could significantly disrupt the morphology of mitochondria, and suppress the migration and invasion of HCC cells. The growth of HCC cells in the animal tumorous model was significantly inhibited after treatment with Vinco(10 mg/kg/day) for 3 days. The results of the present study indicated that Vinco(10-80μg/ml) played a role in activating caspase-3, promoting the expression of Phosphatase and tensin homolog (PTEN), and inhibiting the phosphorylation of AKT(Ser473) and mTOR(Thr2448), Vinco also has a trait for suppressing the expression of C-X-C chemokine receptor type 4 (CXCR4), Src, Matrix metallopeptidase 9 (MMP9), epithelial cell adhesion molecule (EpCAM), Ras, Oct4 and cancer stem cell "stemness markers" CD133 and CD44 in HCC cells.

Conclusions: Vinco has a role in inhibiting the malignant phenotype (behaviors) of HCC cells; the role molecular mechanism of Vinco may be involved in the restraining the expression of the growth-, metastasis-related factors Src, Ras, MMP9, EpCAM, CXCR4, and "stemness markers" CD133 and CD44; and activating the activity of caspase-3 and blocking PI3K/AKT signaling pathway. Thus, Vinco is available for chemotherapy to HCC patients.

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Insilco studies in breast cancer drug design

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Everyone agrees that finding new drugs is a hard, expensive, time-consuming and complicated task. On average, the traditional drug development pipeline takes about 12 years and costs about $2.7 billion to bring a new drug to market. How to lower the cost of research and speed up the process of developing new drugs has become a difficult and important question for the pharmaceutical industry. Computer-aided drug discovery (CADD) has become a powerful and promising way to design drugs that are cheaper, work better and can be made faster. In recent years, the rapid growth of computational tools for drug discovery, such as anticancer therapies, has had a big and positive effect on the design of anticancer drugs and given us new insights into cancer therapy. G protein-coupled receptor 116 (GPR116), which is an orphan adhesion receptor, plays a key role in how eukaryotic cells stick together and move. Since abnormal GPCR expression has been found in many cancers, it can be a matter of interest to find a drug that targets GPCR. Even though the role of GPR116 in the spread of metastasis in triple-negative breast cancer (TNBC) has been studied, no drugs that target GPR116 have yet been found. TNBC is an aggressive type of breast cancer that is not caused by hormones and can happen to young women. Since there is no therapy target receptor for TNBC, GPR116 would be a good choice. Chemotherapy is the only treatment that shows promise for TNBC right now, but these drugs cause chemoresistance. Therefore, newer drug molecules can be identified using CADD approach to overcome this problem.

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Effectiveness of psycho-education intervention programme on coping strategies among Jordanian women diagnosed with breast cancer

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Objective: This study aimed to assess the effectiveness of a psycho-education intervention programme in improving the coping strategies of Jordanian breast cancer patients.

Methods: A double-blinded randomized control trial involving 200 participants between the ages of 20 to 65 years old breast cancer patients was performed. apart from those who refused participation patients with chronic diseases and extreme baseline depression scores were also excluded. the control group received standard care twice a week from the social welfare services team facilitator compared to the intervention group that received additional psycho-education intervention programme (PEIP). the coping strategies were measured using the Brief-COPE inventory consisting of 28 items. it was administered on the second and 12th week of trial. the primary end point was compared between pre- and post-intervention. the effect of the intervention between groups, time, and covariates was measured using the generalized linear mixed model (GLMM) analysis.

Results: The mean (SD) of adaptive coping score among the intervention group increased from 5.63 (1.3) at baseline to 6.42 (1.3) at post-intervention. The mean avoidant coping score was 3.87 (1.1) at baseline but reduced to 3.69 (0.8) post-intervention. GLMM showed that women who received the intervention reported significantly higher usage of the adaptive coping strategies after attending the programme (B=0.921, p <0.001).

c**onclusion:** PEIP significantly improved knowledge of breast cancer patients. thus, this programme may be considered as a part of the healthcare services in Jordan towards improving the adaptive coping strategies among breast cancer patients, which may point towards the potential for these services to increase adaptive coping strategies among patients in Jordan.

Implications: PEIP may be considered as psychosocial intervention in public health and healthcare setting to address rising concerns on quality of care among breast cancer patients.

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