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RESEARCH • INNOVATION • HEALTH

CSR INITIATIVE OF KIMS HOSPITALS™

SYMPOSIUM ON TRANSLATIONAL RESEARCH, GENOMICS & EMERGING TECHNOLOGIES

Date: 30th August, 2025

Venue: KIMS Auditorium, 15th Floor, KIMS Hospitals, Minister Road, Secunderabad, TS, India.

Overview

This event is dedicated to showcasing the journey of research "from bench to bedside", emphasizing how laboratory discoveries translate into better diagnostics, treatments, and patient care. Set in Hyderabad, a city where culture and science have harmoniously coexisted for centuries, the conference invites collaborative efforts that could shape the next chapter of medical progress—one where translational research honors tradition while advancing modern healthcare. With a strong focus on translational science, this conference draws inspiration from Hyderabad's rich legacy of knowledge, innovation, and healing.

Topics

- Practice-informed Research: The Clinician's Role in Translational Research
- Biomedical Devices: Innovation and Impact
- Genetic Research in Patients Care
- Translating Laboratory Discoveries into Practice

Symposium

Join us for an exciting one-day research conference and present your innovative ideas through a poster presentation.

About Us

KIMS Foundation and Research Center (KFRC), the research arm of KIMS Hospitals, is committed to translational research that improves healthcare and serves the community. At KFRC, scientists, clinicians, and research fellows work together on transformative ideas, aimed at making a meaningful impact on people's lives.

Why Choose Us

As part of our commitment to research and innovation, KFRC offers a unique blend of clinical excellence and research expertise to support young researchers. With access to expert guidance, state-of-the-art resources, and collaborative opportunities, KFRC serves as the perfect platform to begin or advance your research journey.

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Chairman & Managing Director
KIMS Hospitals
Presiding Guest

Dr. G. Nageswara Rao
Founder & Chair, LVPEI
Chief Guest

Dr. V. Bhujanga Rao
Chairman of KIMS Foundation &
Research Centre

Dr. Aswin Dalal
Head, Diagnostic Div, CDFD
Keynote Speaker

Speakers

Dr. S. Manimala Rao
Director - Medical Education

Dr. Subash Kaul
Senior Consultant Neurologist

Dr. Manas K Panigrahi
Senior Neurosurgeon

Dr. R.K. Jain
Senior Cardiologist

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Mechanical control of epithelial surveillance: understanding early events in tumorigenesis

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Abstract

Introduction: Epithelial tissues continuously monitor and eliminate aberrant cells to preserve structural integrity and function. This process involves the cells being 'extruded' apically towards the empty lumen, eventually undergoing anoikis, known as Epithelial Defence Against Cancer (EDAC). However, when multiple transformed cells are present within the epithelium, after a certain period following the onset of EDAC, the mutant cells begin to be extruded basally into the extracellular matrix, alongside continued EDAC activity. These basally localized cells may be potential precursors of tumor formation and cancer.

Methods: The methods used include *in vitro* co-culture of wild-type MDCK-WT and MDCK- GFP-HRas^{V12} mutants and intestinal organoids from C57BL/6 mice. We use immuno- (cyto/histo)-chemistry and confocal microscopy for cell and molecular biological studies.

Results: Over time, we have come to understand that this process is governed by multiple mechanical factors, including the properties of the extracellular matrix, overall tissue mechanics, and the mechanical characteristics of intracellular organelles, particularly the cell nucleus. We are now investigating how specific physiological conditions, such as local tissue curvature and fibrosis, influence this process and dictate the direction of cell extrusion.

Conclusion: Understanding how mechanical and physiological factors influence cell extrusion provides key insights into early cancer prevention. In the long term, this knowledge could be translated into therapeutic strategies aimed at reinforcing EDAC mechanisms or blocking basal extrusion pathways using engineered mouse models and human intestinal organoids, ultimately contributing to early intervention and suppression of tumor initiation in epithelial tissues.

Aging promotes inflammation and platelet dysfunction – Tailoring new strategies to prevent age-associated cardiovascular events

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Abstract

Background: Aging is associated with a decline in the function of platelets, thereby increasing the risk for atherothrombosis and myocardial infarction-related mortality. Recent studies have shown that age-associated elevation of pro-inflammatory environment, called "inflammaging" alters the transcriptome and eventually disrupts the function of megakaryocytes, precursor cells that form platelets through a process called "megakaryopoiesis". However, the underlying mechanism through which inflammaging stimuli causes derangement of megakaryocyte function is unknown.

Methods: Briefly, human megakaryoblast cells (MEG-01) were treated with pro- inflammatory cytokines to mimic the inflammaging environment. Following the treatments, the cells were processed for biochemical studies. Further, targeted metabolomics and Seahorse XF Cell Mito Stress tests were performed to measure the levels of cellular metabolic pathway metabolites and mitochondrial function, respectively.

Results: Exposure of the MEG-01 cells to inflammaging stimuli increased differentiation, maturation and cellular senescence. Targeted metabolomics showed inflammaging environment caused unregulated accumulation classical energy metabolism pathway metabolites, leading to a decline in mitochondrial function as assessed by Seahorse Mito Stress tests. However, knock-down of one LncRNA using RNA interference abolished the damaging effects of inflammaging stimuli, causing regulated differentiation, maturation and metabolite load in MEG-01 cells, thereby improving mitochondrial function.

Conclusion: Identification and blocking of the novel LncRNA in the megakaryocytes provide a promising therapeutic strategy to prevent the formation of dysfunctional platelets during aging, thereby decreasing the risk for atherothrombotic-related cardiovascular mortalities.

Testing behavioral and molecular efficacy of vitamin D3 supplementation in a preclinical model of schizophrenia

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Abstract

Background: MK-801 (dizocilpine) mirrors some psychotic symptoms observed in schizophrenia (SCZ). However, less attention has been paid to the anti-psychotic effect of a common neurosteroid, Vitamin D3 (VD), in SCZ. Schizophrenia is a complex mental health disorder that is often associated with glutamatergic hypofunction, which contributes to cognitive and behavioural impairments. Given the limited understanding of how VD modulates these glutamatergic abnormalities and MK-801 induced psychosis, the present study investigated its therapeutic potential in an SCZ mouse model, proposing that VD-VDR nuclear signaling can alleviate glutamatergic hypofunction in SCZ.

Methods: Male C57BL/6 mice (8-10 weeks) were divided randomly into different groups - GR-I-(Control), GR-II (SCZ; MK-801[0.5 mg/kg/day]; i.p injections were performed for two weeks), GR-III (VD only – 500, 1000, 2000 IU/kg/day for two weeks) and GR- IV (**VS** - VD pre supplemented – 500, 1000, 2000 IU/kg/day for two weeks, followed by MK-801 via i.p for an additional 2 weeks). At the end of one month, all mice were decapitated for brain tissue extraction. Various behavioral, biochemical, and gene expression analyses were performed on multiple groups.

Results: Pre-supplementation of VD to SCZ mice (MK-801 induced) decreased hyperlocomotion activity in SCZ mice and rescued the expression of key synaptic protein markers and ionotropic receptor gene expression in the prefrontal cortex.

Conclusion: Early intervention with nutraceuticals like Vitamin D3 (VD) may help in alleviating some symptoms of psychosis in SCZ.

Machine learning–driven, OECD-aligned prediction of HDAC1 inhibition for drug discovery applications

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Abstract

Accurate prediction of bioactivity is critical for accelerating drug discovery while maintaining regulatory compliance and model interpretability. Here, we present a rigorously validated, OECD-compliant machine learning model for predicting the pIC_{50} values of histone deacetylase 1 (HDAC1) inhibitors using an advanced machine learning pipeline for predicting the inhibitory concentration potency (pIC_{50}) of compounds targeting HDAC1, an important enzyme involved in epigenetic regulation and cancer therapy. A large non-redundant dataset of 7,311 unique compounds retrieved from ChEMBL, subjected to rigorous preprocessing to remove duplicates, invalid SMILES, and extreme values. Multiple cheminformatics descriptors were computed, including Morgan, MACCS, Atom Pair, Functional-Class Fingerprints (FCFP), Topological Torsions, 12 basic molecular descriptors, 50 extended physicochemical and fragment-based descriptors, and k-mer based SMILES sequence features. Following outlier removal (IQR method), the feature matrix (1,500 selected variables) was optimized through a three-stage feature selection: variance thresholding, statistical ranking (SelectKBest), and Recursive Feature Elimination using Random Forests. The final model employed a stacking ensemble integrating LightGBM, XGBoost, and Random Forest regressors, with a Ridge regression meta-learner and 7-fold cross-validation. Model evaluation on the test set achieved R^2 = 0.7515, MSE = 0.3866, and MAE = 0.439, with strong Pearson (0.868) and Spearman (0.872) correlations. Residual analysis indicated minimal bias, and over 90% of predictions fell within ±1 pIC_{50} unit. Phytocompounds from the IMPPAT database were screened for Lipinski and ADMET properties, and molecular docking studies were conducted in order to gain structural insights on HDAC1 inhibition.

Modelling tumor heterogeneity in OSCC using 3D patient-derived cultures for evaluating personalized GR-mediated drug delivery

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Abstract

Traditional 2D culture systems fall short in mimicking the complex tumor microenvironment (TME) of Oral Squamous Cell Carcinoma (OSCC), limiting their translational relevance. To address this, we established pre-clinical models of patient-derived organoids (PDOs), and patient-derived cells (PDCs) from oral cancer biopsies. These models reflected patient-specific heterogeneity, including differences in TME components, morphology, growth potential, and migratory or invasive behavior. Conventional anti-cancer treatments such as surgery, chemotherapy, and radiotherapy exhibit setbacks in terms of non- selective killing of cells, higher dosing, inaccessible tumor sites, and higher relapse rates. To address these drawbacks, we aimed to target Glucocorticoid Receptors (GR) of OSCC using a cationic liposomal delivery system to selectively co-deliver the chemotherapeutic drug, Paclitaxel, for the targeted delivery of paclitaxel towards OSCC (D1XP), and evaluated the cytotoxicity of D1XP across the developed pre-clinical models. It was observed that the D1XP significantly enhanced cytotoxicity compared to the non-targeted formulations (D1P) and pristine paclitaxel, with effective tumor reduction in PDOs, even at lower doses. The composition of the TME was observed to vary across patient samples, resulting in patient- specific responses to D1XP concentrations and reinforcing the need for personalized cancer therapies. These findings emphasize the utility of 3D patient-derived models in replicating OSCC TME for preclinical drug testing and underscore the potential of GR-targeted delivery in overcoming the tumor heterogeneity in oral cancer.

METTL3 promotes oral squamous cell carcinoma by regulating miR-146a-5p/SMAD4 axis

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Abstract

Oral squamous cell carcinoma (OSCC) is the most common malignant tumor in the oral cavity. Among various gene regulatory processes influencing OSCC, reversible m6A RNA methylation has emerged as a key player. m6A RNA methylation modulates various facets of cellular RNA metabolism. Frequently m6A regulators i.e. m6A writers, erasers, and readers have been reported to be ectopically expressed in multiple cancers. METTL3, the primary m6A methyl transferase, is significantly upregulated in OSCC cells leading to increased global m6A levels. Interestingly, METTL3/m6A RNA methylation has been reported to positively regulate miRNA biogenesis by modulating the processing of primary miRNAs. However, comprehensive studies exploring the role of m6A-regulated miRNAs in OSCC are sparse. To dissect the role of METTL3-regulated miRNAs in OSCC, small RNA sequencing of OSCC cells upon METTL3 depletion was performed and identified miR-146a-5p as one of the top downregulated candidates. METTL3-depletion/treatment with STM2457, a catalytic inhibitor of METTL3, led to an appreciable accumulation of primary-miR-146a in OSCC cells. Further, to interrogate the role of miR-146a-5p in OSCC, we performed *in vitro* pathophysiological assays to examine cell proliferation, apoptosis, colony formation, migration, and invasion of OSCC, upon its inhibition or overexpression. Our data conclusively points to its oncogenic role in OSCC cells. Bioinformatic analysis identified SMAD4, a well-known tumor suppressor gene, as one of the targets of miR-146a-5p, and the direct interaction between miR-146a-5p and SMAD4 3'UTR was confirmed by luciferase reporter assay. Taken together, our study uncovers a novel role for METTL3/miR-146-5p axis in regulating OSCC progression by targeting SMAD4.

Integrative omics analysis to discover chromatin regulators of cancer-induced muscle atrophy

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Abstract

Cancer cachexia (CC) is a paraneoplastic syndrome exhibiting unintended and progressive loss of muscle mass due to protein hypercatabolism. Though CC accounts for ~30% of cancer deaths, there is no single drug available for cachexia treatment. In the absence of somatic mutations, pro-inflammatory cytokines secreted by tumors and aberrant signalling pathways are driving factors for anomalous expression of muscle atrophy-related genes (atrogenes). It is well-known that environmental stimuli trigger biochemical changes in the epigenome that result in remodelling of chromatin structure and culminate in gene expression changes. Therefore, we hypothesize that tumor-derived factors bring epigenetic modifications that drive transcriptional reprogramming in skeletal muscles and upregulation of atrogenes, leading to muscle atrophy. To establish the role of epigenetics in CC, we analyzed 30 publicly available RNA-seq datasets from normal and cachectic skeletal muscles. Gene set enrichment analysis revealed that genes belonging to chromatin, histone, and epigenetic-related processes are differentially regulated in CC. Additionally, Landscape In Silico deletion Analysis (LISA) predicted that many chromatin and epigenetic modulators are involved in differential gene regulation in CC. In an attempt to understand the role of *cis*-regulatory elements in transcriptional regulation, we studied the binding profile of BRD4, an epigenomic reader of enhancers, in normal versus cachectic skeletal muscles. Our analysis uncovered the critical role of variant enhancers in controlling the expression of many atrogenes, including *Trim63* and *Fbxo32*. The fact that cachectic genes rely on these variant enhancers for their expression, targeting their enhancer dependency could be exploited for therapeutic purposes to treat CC.

Precision multimodal diagnostics with mobileimaging and hierarchical analysis

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Abstract

A cutting-edge multimodal imaging application, built on the open-source MedImageInsight vision-language model, introduces an innovative approach to medical diagnostics through a conditional hierarchical classification methodology. This application delivers accurate predictions and detailed diagnoses across diverse modalities, including ultrasound, CT, and histopathology for fatty liver detection, chest X-ray for pneumonia and related conditions, mammography for breast cancer screening, and fundus photography for diabetic retinopathy prediction. Notably, it enables precise analysis of JPG or PNG images captured from mobile devices, enhancing accessibility in varied clinical environments. A GPT-2 decoder head generates comprehensive diagnostic reports post-prediction, improving clinical utility. Performance evaluations, as reported by the model's developers under specific testing conditions, demonstrate Area Under the Curve (AUC) scores ranging from 80% to over 90% across modalities, reflecting robust diagnostic reliability. Developed in Lightning AI Studios, the application leverages GPU acceleration to achieve inference times under 2 seconds, ensuring minimal latency for efficient clinical workflows. This high-accuracy, low-latency system offers a scalable solution for healthcare providers, facilitating timely and precise diagnoses across multiple imaging tasks. Its open-source framework encourages collaborative innovation, positioning it as a transformative tool in precision medicine. This research underscores the potential of integrating advanced vision-language models with hierarchical classification and mobile imaging capabilities to address complex diagnostic challenges, paving the way for enhanced patient outcomes in multimodal medical imaging.

Revolutionizing early hepatocellular carcinoma detection: A six-gene signature powered by machine learning

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Abstract

Hepatocellular carcinoma (HCC), a leading cause of cancer mortality worldwide, is often diagnosed at advanced stages, limiting effective interventions. Current diagnostics, including liver function tests (LFTs), alpha-fetoprotein (AFP) panels, and ultrasound, lack specificity, necessitating precise biomarkers for early detection. We integrated RNA-sequencing data from GEO, UCSC Xena, and GREIN, employing advanced Machine Learning (ML) feature selection techniques, including Recursive Feature Elimination (RFE) and Random Forest Importance (RFI), to identify a robust six-gene signature: CDKN3, LIFR, MKI67, TOP2A, SLC5A1, and VIPR1, validated across diverse Japanese, American, Asian, and African cohorts. A Random Forest (RF) model using 52 significant genes (SGs) achieved an accuracy of 96% and an Area Under the Receiver Operating Characteristic curve (AUC-ROC) of 0.99 on an independent test set, while a logistic regression-based risk score leveraging the six-gene panel yielded an accuracy of 94% and an AUC-ROC of 1.00, demonstrating exceptional predictive power for distinguishing HCC from normal tissue. Functional enrichment analyses via Gene Ontology, DAVID, and STRING revealed associations with critical HCC-related processes, including cell cycle regulation, apoptosis, and extracellular matrix remodeling. Kaplan-Meier survival analysis indicated that upregulated CDKN3, TOP2A, and MKI67 correlate with poor prognosis, while downregulated LIFR, SLC5A1, and VIPR1 are linked to better survival outcomes. Validated by the Human Protein Atlas (HPA) and Comparative Toxicogenomics Database (CTD), this six-gene signature offers a promising non-invasive diagnostic tool for early HCC detection. These findings lay a foundation for targeted diagnostics and personalized therapeutic strategies, advancing precision oncology for improved HCC management.

Interpretable prediction of drug efficacy in breast cancer using integrated genomic and chemical profiles

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Abstract

Background: Accurate prediction of drug sensitivity in cancer cell lines is crucial for precision oncology and patient-specific therapies. Many existing computational approaches fail to integrate multi-modal biological and chemical features and struggle with high-dimensional, imbalanced pharmacogenomic data, limiting predictive accuracy and interpretability.

Methods: We developed a machine learning framework combining pharmacogenomic profiles (mutation status, copy number alterations, microsatellite instability) with molecular fingerprints and descriptors for 85 anti-cancer drugs generated via PaDEL from SMILES strings. Data from 40 breast cancer cell lines in the Genomics of Drug Sensitivity in Cancer (GDSC) dataset were used. A three-stage feature selection process (Boruta, mRMR, XGBoost) reduced drug feature dimensionality while retaining 130 cell line features. Models—including Random Forest, Logistic Regression, KNN, Naïve Bayes, tuned LightGBM, and XGBoost—were trained, with LightGBM evaluated using 3-fold cross-validation, class weighting (class_weight='balanced'), and grid search to address imbalance (233 sensitive vs. 3167 resistant samples).

Results: LightGBM achieved AUROC = 0.9455, AUPRC = 0.5148, Accuracy = 0.8415, F1-score = 0.4481, Recall = 0.9409, MCC = 0.4732, demonstrating suitability for sparse biomedical datasets. Shapley Additive Explanations (SHAP) identified cnaBRCA25 (not mutated) as a consistent resistance marker and cnaBRCA47 (mutated) as a context-dependent biomarker. These BRCA-related features are functionally important due to their role in DNA repair pathways, influencing drug response in breast cancer.

Conclusion: The framework integrates diverse data modalities, manages severe class imbalance, and offers interpretable biomarker insights. While promising, the study is limited by dataset size; further validation on larger, independent cohorts is needed for clinical translation.

Tumor-suppressive role of m6A eraser proteins FTO and ALKBH5 in oral squamous cell carcinoma

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Abstract

Oral squamous cell carcinoma (OSCC), the most common oral malignancy, is influenced by various gene regulatory processes. Among the various epigenetic modifications, N6-methyladenosine (m6A RNA methylation) has recently emerged as a major research interest. m6A RNA methylation, a key internal and reversible RNA modification, plays a crucial role in cancer development and progression by regulating various facets of RNA metabolism. Among m6A-modifying enzymes, the m6A erasers (m6A demethylases) fat mass and obesity associated protein (FTO) and Alkb homolog 5 (ALKBH5) are frequently dysregulated in various cancer types. In this study, we investigated the biological significance of FTO and ALKBH5 in OSCC via loss-of-function studies coupled with in vitro pathophysiological assays. Our results demonstrate significantly reduced transcript and protein levels of FTO and ALKBH5 in OSCC cells compared to control normal esophageal epithelial cells. Furthermore, siRNA-mediated transient knockdown of FTO and ALKBH5 enhanced cell viability, colony formation, migration, and invasion while decreasing apoptosis in OSCC cells. Notably, RT-qPCR analysis revealed upregulated expression of integrin alpha 6 (ITGA6) and apolipoprotein (APOE) mRNAs, two known m6A-regulated transcripts, upon depletion of FTO or ALKBH5. These findings highlight the tumor-suppressive function of FTO and ALKBH5 in OSCC and underscore their potential as prognostic biomarkers and therapeutic targets in this malignancy.

Machine learning integrated qsar and molecular docking for identifying natural product derived PDE4B inhibitors in psoriatic arthritis

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Psoriatic arthritis (PsA) is a chronic immune-mediated inflammatory arthropathy causing joint pain, stiffness and progressive structural damage often accompanied by enthesitis, dactylitis and reduced mobility. Left untreated, it can result in irreversible disability and a significantly diminished quality of life. Current management strategies include non- steroidal anti-inflammatory drugs (NSAIDs), corticosteroids and conventional and biologic disease modifying antirheumatic drugs (DMARDs). Among these, phosphodiesterase 4 (PDE4) inhibitors are an important oral, non-biologic option. However, approved agents such as Apremilast are associated with side effects including gastrointestinal disturbances, headache, weight loss and depression. Bioactivity data for PDE4B inhibitors were obtained from ChEMBL and transformed into readable molecular descriptors. Feature refinement using correlation filtering and importance ranking yielded 32 optimal features for model development. A predictive model for IC50 values against PDE4B was built using these features achieving an R^2 of 0.93. The model demonstrated strong predictive performance and robustness on a large and diverse dataset confirmed by 10-fold cross-validation. KMeans clustering was used to identify high-performing molecular subsets from which representative scaffolds were extracted. These scaffolds were screened against over 400,000 natural products and compounds with predicted IC50 values below 1 µM were prioritized for molecular docking. Docking analysis identified a plant-derived xanthone with high binding affinity and key interactions comparable to approved PDE4 inhibitors supporting its potential as a promising lead for further preclinical evaluation.

Effectiveness of a structured teaching programme on knowledge regarding text neck syndrome among degree students

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Introduction: Text Neck Syndrome is characterized by increased curvature of the cervical spine, resulting from the prolonged forward tilt of the head while using electronic devices. This is a problem that comes hand in hand with technology. A study to assess the effectiveness of STP on knowledge regarding Text Neck Syndrome among degree students.

Methodology: A quasi-experimental study on group pretest and posttest design was chosen for this study. The study was carried out among 40 degree student studying at Wesley College, Secunderabad, Telangana selected by random sampling technique using structured knowledge questionnaires. The pretest level of knowledge was assessed using structured knowledge questionnaire are structured teaching program was given. Post test was conducted after 7 days.

Results: Study finding revealed that the majority (52.5%) had adequate knowledge (47.5%) moderately adequate knowledge, and none of them had inadequate knowledge regarding text neck syndrome. The mean posttest knowledge score 14.3 with SD 3.0 was significantly higher than the pretest means score 6.5 with SD

2.26 with a mean difference of 7.8. Since the calculated 't' value 17.05 which was greater than the table value (3.55) with degree of freedom 39 at $p<0.001$ level of significance.

The pretest knowledge scores of degree students and the demographic variables like age, gender type of gadget using duration of using hand held device and previous knowledge regarding test neck syndrome were found to be non-significant.

Conclusion: STP was found to be effective in improving knowledge of degree students regarding test neck syndrome by the scores of the posttest as it is evident.

Edible probiotic prototype yogurt vaccine for COVID-19

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Abstract

Introduction: The administration of COVID-19 vaccines through injections posed challenges during the pandemic due to pain, the need for clinical supervision, and limited compliance. As a potential alternative, an edible probiotic yogurt-based vaccine (YoVac) was conceptualized. We hypothesized that YoVac prepared using Lactobacillus carrying an antigen-coding gene (donor) can transfer the same to other bacteria (recipients) in the human gut microbiome through lateral gene transfer for boosted antigen levels, potentially triggering a robust immune response.

Methods: YoVac uses Lactobacillus engineered with an antigen-coding gene, which can be transferred to other gut bacteria through lateral gene transfer (LGT).

Results: In vitro experiments confirmed successful lateral gene transfer (LGT) of pRBD-Ampr from Lactobacillus (donor) to E. coli and Helicobacter pylori (recipients), as evidenced by both acquisition of ampicillin resistance and RBD protein expression, resulting in enhanced antigen production with the potential to strengthen the immune response.

Conclusion: This proof-of-concept study demonstrates that probiotic Lactobacillus can serve as a gene-delivery vehicle for antigen expression and transfer to other bacterial species. YoVac was developed using Lactobacillus carrying a recombinant plasmid (pRBD-Ampr) encoding the SARS-CoV-2 spike receptor-binding domain (RBD) gene and an ampicillin-resistance marker.

A descriptive study to assess the Empty Nest Syndrome among the parents of B.Sc Nursing students studying at selected Nursing Colleges, Hyderabad, Telangana

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Abstract

Introduction: The Empty Nest Syndrome (ENS) has been a term used in psychology to denote the psychological symptoms that arise when the youngest child leaves the family for work or further studies causing a void in the family leaving the aged parents behind is termed as the empty nest. It is a term used to describe the long-lasting maladaptive responses exhibited by parents once their last child moves out of their household and thereby leaves the parents alone at home. To assess the knowledge regarding Empty Nest Syndrome among parents of B.Sc Nursing students.

Methodology: The research design adopted for the study was descriptive research design. Convenient sampling technique was used to select the sample to assess the knowledge regarding Empty Nest Syndrome among the parents of B.Sc Nursing students.

Results: The major finding of the study reveals that the main knowledge scores 48.9 with SD 8.12. It reveals that the cal 't' value for knowledge 167-250 was greater than the tabulated 't' value with degree of freedom 60 at 0.05 level of significant. The research design adopted for this study is Descriptive design approach. One group pretest and post-test pre-experimental study was conducted to assess the knowledge regarding Empty Nest Syndrome. The pilot study was conducted in JMJ College of Nursing, Erragadda, Secunderabad, Telangana. Statistically it is found that there is high significant association between Age in years, education, family monthly income regarding Empty Nest Syndrome among parents.

A novel multi-epitope vaccine design against *Streptococcus pneumonia*

Yogeshwar Devarakonda[1]

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Abstract

Streptococcus pneumoniae a leading cause of sepsis, meningitis, and pneumonia remains a significant health threat particularly in children and immunocompromised adults. Existing vaccines have limited serotype coverage, and the rise of non-vaccine serotypes further poses an ongoing challenge to global health. In this study, a multi-epitope subunit vaccine targeting four key surface proteins Ply, PsaA, PspA, and PspK was developed using immuno-informatics tools. B-cell and T-cell epitopes were predicted via NetCTL, IEDB, and ABCpred databases. The vaccine construct was evaluated for antigenicity, allergenicity, toxicity, and physicochemical properties. Its structure was refined and validated, followed by docking with TLR-4 using ClusPro. The vaccine construct was cloned into pET-28a vector and expressed in Escherichia coli. Successful overexpression of the protein was achieved, and its purification was confirmed through SDS-PAGE and Ni-NTA affinity chromatography. The circular dichroism spectroscopy analysis indicated a stable and well-structured conformation of the multiepitope vaccine, while hemolysis assay demonstrated minimal RBC toxicity suggesting the vaccine's reliability and safety. The BALB/c mice was administered with the potential vaccine candidate and immune response was evaluated. The antibody response has been confirmed by western blot. It could potentially activate both humoral and cellular immune responses and has the potential to be a vaccine candidate against S. pneumoniae.

CRISPR-based diagnostic kit for the detection of latent HIV-1 Infections using patient blood samples

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Abstract:

Background: HIV/AIDS continues to be a significant global health challenge, even with the widespread use of antiretroviral therapy (ART). A major obstacle is the persistence of latent HIV-1 reservoirs that remain hidden in the body and cannot be eliminated by ART, creating the risk of viral rebound. Conventional diagnostic techniques such as RT-PCR, ELISA, and viral outgrowth assays (VOAs) have notable limitations in detecting these latent infections.

The present disclosure introduces a CRISPR-based diagnostic kit (CHIKit-SA) designed to detect latent HIV-1 infections using patient blood samples.

Methods: The kit includes a genomic DNA extraction, a CRISPR-Cas9 platform, and an agarose gel electrophoresis setup.

Results: We targeted HIV-1 DNA integrated with in the host Genome. This system provides accurate identification of latent reservoirs and infected host cells.

Conclusion: Importantly, the kit enables early detection of individuals with latent infections, facilitating informed treatment decisions, monitoring of Highly Active Antiretroviral Therapy (HAART) effectiveness, and helps prevent both viral rebound and transmission. Additionally, the kit can trigger reactivation of latent virus, thereby enhancing strategies aimed at more effective treat HIV-1 infection. Indian patents filed: IN202341074827.

RM-3-22: a hydroxamic acid derivative based on TAZQ with anticancer properties that target NSCLC both *in vitro* and *in vivo*

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Abstract

Lung cancer remains the leading cause of cancer-related mortality worldwide, highlighting the urgent need for novel and effective therapeutic strategies. In this study, we developed RM-3-22, a hydroxamic acid derivative based on the TAZQ scaffold, with potent histone deacetylase (HDAC) inhibitory properties, and evaluated its anticancer activity in non-small cell lung cancer (NSCLC) using A549 adenocarcinoma cells as the primary model. The cytotoxic effects of RM-3-22 were investigated using both 2D and 3D culture systems, and its impact on cell viability was assessed via MTT assay. Various fluorescence staining techniques were employed to evaluate autophagy and nuclear morphology. Flow cytometry revealed that RM-3-22 induced apoptosis, mitochondrial membrane depolarization, and G2/M phase arrest. Mechanistic investigations demonstrated that RM-3-22 suppressed the PI3K/Akt/mTOR signaling pathway, thereby inducing autophagy, which in turn contributed to apoptosis and cell cycle arrest. Gene and protein expression analyses via RT-PCR, Western blotting, and immunofluorescence further confirmed these molecular alterations. Functional studies with siRNA-mediated knockdown elucidated the regulatory role of autophagy in RM-3-22-mediated anticancer effects. Notably, RM-3-22 upregulated FTH1, a tumor suppressor gene, and exhibited minimal cytotoxicity in normal cells, suggesting high selectivity. In vivo efficacy was validated using an NOD/SCID xenograft mouse model, where RM-3-22 significantly inhibited tumor growth without apparent toxicity. Overall, RM-3-22 exerts its anticancer effects through multi-faceted mechanisms involving autophagy activation, apoptosis induction, and cell cycle arrest, positioning it as a promising therapeutic candidate for the treatment of NSCLC.