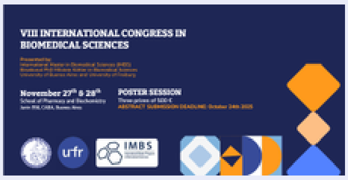

Wednesday, October 29, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

Generation of a PSEN1/PSEN2 Double Knockout HEK293T Line Using CRISPR/Cas9 for Translational Studies in Alzheimer's Disease

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THE POSTER APPLIES FOR PRIZE

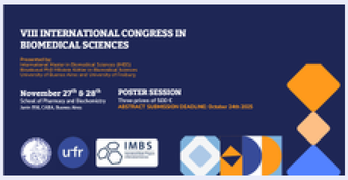
NO

Terms and Conditions

Accepted

POSTER ABSTRACT (350 WORDS MAXIMUM)

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by memory loss and cognitive decline. While most cases are late-onset, early-onset AD (EOAD) accounts for a smaller proportion and often exhibits autosomal dominant inheritance. Genetic studies have identified pathogenic variants in APP, PSEN1, and PSEN2 as the main causes of EOAD. PSEN1 and PSEN2 encode catalytic components of the γ-secretase complex, which mediates APP cleavage and generation of soluble Aβ peptides. Disruption of this process leads to Aβ accumulation and formation of amyloid plaques, a pathological hallmark of AD. To investigate the impact of presenilin loss on APP processing, we generated a double knockout (KO) cell line for PSEN1 and PSEN2 using CRISPR/Cas9 genome editing. The parental HEK293T line, previously knocked out for PSEN1, was transfected by lipofection with a CRISPR/Cas9 expression plasmid targeting PSEN2. Cells were cultured in DMEM supplemented with 10% fetal bovine serum (FBS) under standard conditions. Guide RNAs were designed to target an early exon of PSEN2, minimizing potential off-target sites. Transfected cells were selected by puromycin treatment, and single-cell clones were isolated by limiting dilution and expanded. Genomic validation was performed by Sanger sequencing, confirming the presence of indels at the targeted PSEN2 locus. Loss of Presenilin-2 protein expression was assessed by Western blot, which verified the absence of PSEN2 bands in the double KO clones compared with wild-type controls, confirming the successful generation of a stable double KO line. This cellular model enables the study of the combined loss of presenilin function in APP metabolism, representing a valuable translational tool for exploring Alzheimer's disease mechanisms and testing therapeutic strategies targeting γ-secretase–related pathways.



Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

From Reactive to Regenerative: IMT504 Unlocks the Pro-Remyelinating Potential of Astrocytes

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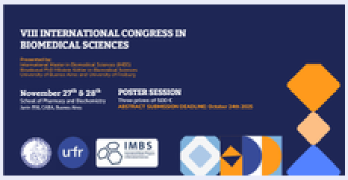
POSTER ABSTRACT (350 WORDS MAXIMUM)

Many central nervous system (CNS) diseases involve demyelination, a pathological process characterized by the loss of myelin sheaths around axons. The regeneration of these sheaths, known as remyelination, is crucial for restoring axonal function. While successful remyelination generally depends on oligodendrocyte progenitor cell (OPC) proliferation and differentiation, other cell types also play critical roles. For instance, recent studies have highlighted beneficial and detrimental roles of astrocytes in remyelination.
IMT504 is a non-CpG oligodeoxynucleotide comprising 24 nucleotides and characterized by two specific PyNTTTTGT motifs. Its safety profile has been established through pharmacokinetic and toxicological

studies, and a phase I clinical trial has been recently approved (Resol. DI-2023-6969-APN-ANMAT#MS). Building on previous work (Mathieu et al., 2024) which demonstrated its benefits in neuroinflammation and oligodendrogenesis, this study investigates how IMT504 influences astrocyte function.
Primary astrocyte cultures were obtained from cerebral cortical tissue of 1- to 2-day-old rats and used to analyze the direct effects of IMT504. Cells were treated with or without IMT504 and fixed for immunocytochemistry or harvested at different times for Western blot or qPCR analyses. For phagocytosis assays, astrocytes were treated with IMT504 or saline solution for 24 h and subsequently incubated for 1 h with fluorescent latex beads. BrdU was added to the cultures for 24 h and fixed for proliferation evaluation. Results show that IMT504 was incorporated into cells and inhibited astrocyte proliferation. Furthermore, IMT504 produced morphological changes and reduced astrocyte migratory capacity. It also modulated phagocytic activity, increased GFAP and BDNF protein levels, and shifted gene expression toward an anti-inflammatory phenotype by acting on different signaling pathways.
These findings suggest that IMT504 could enhance CNS remyelination capacity by modulating astrocyte function, highlighting its therapeutic potential for demyelinating disorders such as multiple sclerosis.



Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

TOWARDS NON-INVASIVE GOLD NANOPARTICLE-BASED THERAPIES FOR CENTRAL NERVOUS SYSTEM DISEASES

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Terms and Conditions

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POSTER ABSTRACT (350 WORDS MAXIMUM)

In this study, we synthesized and characterized gold nanoparticles (GNPs) with potential therapeutic applications for central nervous system (CNS) disorders. GNPs were prepared using linear polyethyleneimine (PEI) as both a reducing and stabilizing agent. Their physicochemical properties were analyzed using various spectroscopic and microscopic techniques, confirming their nanoscale size, stability, and spherical morphology.
The biocompatibility of GNPs was evaluated through cytotoxicity assays in various neural cell lines—N2A (neuroblastoma), BV2 (microglia), and OLN93 (oligodendrocytes)—as well as in primary cultures of neural stem cells (NSCs) and microglia. Results demonstrated that GNPs exhibited no cytotoxic effects at the tested concentrations, maintaining high cell viability across all models. The internalization of fluorescently labeled nanoparticles was confirmed by confocal microscopy, showing efficient uptake in all tested cell types.
In primary NSC cultures, GNP treatment promoted differentiation toward the oligodendroglial lineage. This was evidenced by a higher percentage of myelin basic protein (MBP)-positive cells compared to controls, together with increased morphological complexity, suggesting enhanced maturation and process extension of oligodendrocytes. These findings indicate a pro-differentiation effect of PEI-stabilized GNPs on NSCs.

In primary microglial cultures, GNP exposure did not compromise viability and significantly increased phagocytic activity, a key function in debris clearance during remyelination.
Interesting, we found that GNP strongly prevents ROS upregulation and nitrite production in LPS-treated microglia, even though GNP alone produces a non-significant increase in ROS and nitrite levels. These findings suggest that GNPs exert an immunomodulatory effect, preserving microglial homeostasis and promoting a reparative phenotype.
Finally, in vivo administration of GNPs via intranasal or intraperitoneal routes was performed. Gold biodistribution quantified by Inductively Coupled Plasma Mass Spectrometry (ICP-MS) revealed significant accumulation in CNS regions following intranasal delivery, supporting the feasibility of non-invasive administration.
Overall, PEI-stabilized GNPs exhibited high biocompatibility, efficient cellular uptake, promote oligodendrocyte differentiation on NSCs, and enhance microglial phagocytic capacity while modulating inflammatory responses. These findings highlight their potential as a versatile and minimally invasive platform for future neuroregenerative and drug-delivery strategies.

Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

Role of the Oligodeoxynucleotide IMT504 in Cortical Remyelination Following Experimental Demyelination in Rats

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THE POSTER APPLIES FOR PRIZE

NO

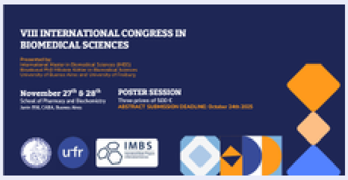
Terms and Conditions

Accepted

POSTER ABSTRACT (350 WORDS MAXIMUM)

Demyelination is a pathological process characterized by myelin loss from around axons, while remyelination is the repair response which restores myelin and resolves functional deficits. Multiple sclerosis is a high-incidence inflammatory demyelinating disease in which remyelination frequently fails. IMT504 (IMT) is a non-CpG oligodeoxynucleotide consisting of 24 nucleotides and characterized by 2 specific PyNTTTTGT sequences. On the basis of IMT immunomodulatory effects and regenerative properties, and our previous results showing its beneficial effects on neuroinflammation and remyelination in the corpus callosum of cuprizone (CPZ)-demyelinated rats, this work aims to study IMT role in microglial and oligodendrocyte (OL) cell populations in the cerebral cortex (Ctx). We subcutaneously administered IMT every day for five days before CPZ withdrawal. Brain samples were then analyzed 0 (T0), 3 (T3), 7 (T7), and 10 (T10) days after CPZ withdrawal. For each experimental time point, samples were processed in parallel: coronal sections were used for immunohistochemical analysis, and

Ctx homogenates were prepared to assess signaling pathways by Western blot. Immunohistochemical analyses showed that IMT did not modify the population of Iba1+ microglial cells or APC+/Sox10+ OLs at any of the time points evaluated. However, IMT induced a significant increase in PDGFRα+ OL progenitor cells (OPCs) at T3 and MAG+ mature OLs at T7, as compared to CPZ-saline-treated animals. Western blot results revealed a significant increase in ERK1/2 phosphorylation in the Ctx of CPZ–IMT rats at T0. ERK1/2 signaling activation is associated with increased myelin sheath thickness and repair processes following neuronal injury. In contrast, a decrease in p38 phosphorylation was detected at T3, which may reflect a regulatory mechanism that initially promotes progenitor expansion and allows the ERK1/2 signaling pathway to drive maturation, as evidenced by a later increase in mature OLs. These findings support the potentially beneficial properties of IMT in the remyelination process of demyelinated cortical lesions.



Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA Cellular & Molecular Biology

POSTER TITLE THE PRO-INFLAMMATORY EFFECT OF HISTAMINE H1 RECEPTOR ACTIVATION IS REDUCED BY THE ANGIOTENSIN-(1–7) MAS RECEPTOR

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THE POSTER APPLIES FOR PRIZE YES (I understand that if my poster is pre-selected I will have to give a 5 minute oral presentation to the congress audience for final decision).

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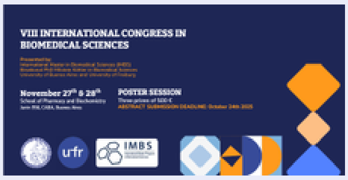
POSTER ABSTRACT (350 WORDS MAXIMUM)

Arterial hypertension is one of the major health problems in modern society. Alterations in the sympathetic and parasympathetic nervous systems, the renin-angiotensin system (RAS), genetic predisposition, environmental factors, age, and inflammation all contribute to its development. The protective axis of the RAS, composed of angiotensin (Ang) (1-7) and the Mas receptor (MasR), exerts vasodilatory, hypotensive, antifibrotic, and anti-inflammatory effects that counteract the actions of the pressor axis of the system. The anti-inflammatory effects of the Ang-(1-7)/MasR axis have been demonstrated in several pathological conditions, including stroke, atherosclerosis, neurodegenerative diseases, pulmonary fibrosis, and asthma. In contrast, histamine, through the type 1 receptor (H1R), induces inflammatory responses. Both MasR and H1R belong to the G protein-coupled receptor (GPCR) family. GPCRs have been shown to form heterodimers that modulate receptor-mediated signaling. Considering that MasR and H1R mediate opposite effects in inflammation, we investigated the response triggered by the stimulation of these receptors in human macrophages exposed to a pro-inflammatory stimulus, and whether such response is influenced by cross-talk between them. H1R stimulation increased interleukin (IL)-8 and IL-1β expression. MasR blockade further enhanced IL-8 secretion induced by a specific H1R agonist, but did not modify IL-1β levels, suggesting cross-talk between both receptors. The interaction between H1R and MasR was confirmed by FRET analysis in HEK293T cells coexpressing both receptors. H1R-MasR heteromerization was not affected by any of the receptor agonists or antagonists, indicating that it is constitutive. In

HEK293T cells transfected with H1R, the specific H1R agonist increased IL-6 promoter activity, an effect that was absent in cells co-transfected with H1R and MasR. Overall, these results demonstrate a cross-talk between H1R and MasR in inflammation, with MasR counterbalancing the pro-inflammatory effect of H1R.



Tuesday, October 21, 2025

VIII International Congress in Translational Medicine

AREA
Cellular & Molecular Biology

POSTER TITLE
GLIAL RESPONSE AFTER COLD INJURY IN MICE: "INVOLVEMENT OF THE C3-C3aR PATHWAY"

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Terms and Conditions
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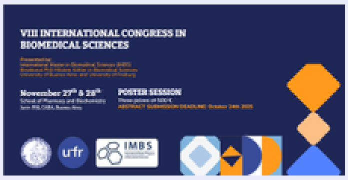
POSTER ABSTRACT (350 WORDS MAXIMUM)

During reactive gliosis, astrocytes respond with an increase in size and morphological complexity, transcriptional changes, loss of homeostatic functions and gain of pro-inflammatory features (among others). Understanding astrocyte reactivity has become of main interest to understanding and modulating injury progression.
We hypothesize that cold injury, an experimental model of brain edema, results suitable for conducting studies on progressive reactive astrogliosis.
We here aimed to characterize astrocyte response using an injury model of cold injury in young adult female and male mice (C57/4-5 months) for which we addressed tissue response at 1, 3 and 7 days post lesion (DPL).
Using Immunofluorescent co-labeling followed by epifluorescence and confocal microscopy we observed a progressive increase in GFAP (glial fibrillary acidic protein) immunoreactivity and an increase in AQP4 (aquaporin 4) immunoreactivity at 1DPL. Immunoreactivity of IBA1 (Ionized calcium-binding adaptor molecule 1) progressively increased at 1, 3 and 7DPL while NeuN radically decreased around the injury core. NG2 immunoreactivity addressed at 1DPL, was also increased. We observed an increase in C3

(complement 3) immunoreactivity but with no colocalization with neither GFAP, IBA1 nor NG2 cells. Using qPCR and immunofluorescence we observed an increase in C3aR expression after injury.
We conclude that this model of brain edema promotes a progressive astroglial/microglial response concomitantly with neuronal stress. Further, components of the C3-C3aR pathway were found to be present suggesting a role in injury progression. It is of note that this model will allow addressing cellular response in a context of brain edema, a pathological condition of high clinical relevance.



Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

Characterization of sex-differences in the cortical microglial response to serotonin exposure in an experimental model of neurodevelopmental synaptopathy

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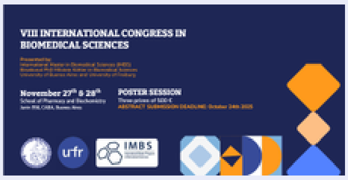
POSTER ABSTRACT (350 WORDS MAXIMUM)

Hyperserotonemia and neuroinflammation are both involved in the neurobiology of autism spectrum disorder (ASD), a known neurodevelopmental synaptopathy. The prenatal valproic acid (VPA) administration in rats (450 mg/kg, i.p. at E10.5) is a well-accepted ASD experimental model, that has been poorly studied in females. Microglia have been reported to mediate neuroinflammation in the VPA model. Interestingly, microglial cells express serotonin receptors. In this study, we aimed to characterize the sex differences of cortical microglia morphological profile and their response to serotonin exposure in the VPA model. We isolated cortical microglia from male and female Wistar rat pups; cells were incubated overnight with different serotonin concentrations (1 μM, 20 μM and 50 μM) and immunostained with Iba-1 to analyse their morphological profiles (reactive: non-ramified: type 1-circumscribed and type 2-"fried egg"; non-reactive: type 3-ramified). Microglia isolated from both male and female VPA rats showed increased basal reactivity compared to those from control animals. In response to serotonin treatment, microglia from control females showed a complex dose-dependent response while microglia from control males decreased their reactivity. Microglia from both VPA males and females remained reactive as in basal conditions when exposed to serotonin. These results reveal there is a sex-dependent response to serotonin in control microglia. On the contrary, microglia from VPA rats failed to respond. This suggests that prenatal VPA exposure disrupts the mechanisms underlying serotonin action on microglia, potentially

contributing to the neuroinflammation observed in this model.



Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

The DNA repair protein TDP1 is relocated to heterochromatin to safeguard the genome in response to DNA damage triggered by Etoposide

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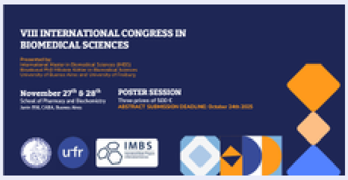
POSTER ABSTRACT (350 WORDS MAXIMUM)

Tyrosyl-DNA-phosphodiesterase 1 (TDP1) overexpression is observed in numerous types of cancer cells, including rhabdomyosarcoma, colon, stomach, pancreas, and endocervical adenocarcinomas. Due to its role in DNA repair, it represents an interesting molecular target for the development new cancer therapies. TDP1 specializes in removing protein adducts from DNA at both 3' and 5' ends, such as cleavage complexes induced by Topoisomerase I and II poisons, respectively. By decreasing heterochromatin regions through chromatin decondensation, histone deacetylase inhibitors (HDIs) increase the cytotoxicity of these poisons. We previously showed that the HDI Trichostatine A sensitizes tumor cells to the chemotherapeutic agent Etoposide (ETO), a Topoisomerase II poison. Furthermore, cell lines overexpressing TDP1 are more sensitive to HDIs. Based on these observations, we decided to focus on studying the relationship between TDP1 and chromatin sub-compartments. In the human cervical adenocarcinoma cell line HeLa, we performed double immunofluorescence labelling of TDP1 and either HP1α, RNA polymerase II (RNPII) or acetyl-K9/14H3 (AcK9/14H3), after treatment with ETO. Our results show that TDP1 localization significantly increases within DNA regions enriched in the heterochromatin marker HP1α, as measured by the threshold overlap score (TOS) and Manders' colocalization coefficient

M1. To corroborate these findings, we performed chromatin immunoprecipitation of TDP1-associated DNA followed by quantitative PCR targeting alpha satellite DNA, which is abundant in centromeric and pericentromeric heterochromatin. Consistently, we observed a significant increase in TDP1 association with alphoid regions after ETO treatment. In line with these results, ETO reduced TDP1 colocalization with AcK9/14H3 and RNPII, both markers of actively transcribed chromatin. The relocation to heterochromatin was related to a DNA repair activity of TDP1, as cells lacking TDP1 accumulated unresolved DNA double strand breaks (γH2AX foci) induced by ETO within heterochromatin, compared to TDP1-proficient cells. Together, these results demonstrate that TDP1 relocates from euchromatin to heterochromatin in response to DNA damage induced by ETO, where it exhibits functional activity. Therefore, our data suggest that the increased toxicity generated by HDIs in combination with ETO could be related to a failure of TDP1 to relocate due to the diminished amount of heterochromatic regions in the DNA.



Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Microbiology and Immunology

POSTER TITLE

MICROFLUIDIC PRODUCTION OF UV-CROSSLINKABLE HYDROGEL BEADS FOR YEAST ENCAPSULATION: A PLATFORM FOR CELL STORAGE AND FERMENTATIVE BIOREACTORS

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims
Traditional cell immobilization methods struggle with low reproducibility, size heterogeneity, and limited mechanical stability. To address these issues for optimal bioprocesses, this study introduces a microfluidic platform for producing UV-crosslinkable poly(ethylene glycol) diacrylate (PEGDA) hydrogel beads. Our main objective was to efficiently encapsulate Saccharomyces cerevisiae yeast, evaluating the beads for long-term storage and use in fermentative bioreactors, emphasizing low final product turbidity.

Methods
The microfluidic setup generated yeast-in-PEGDA hydrogel beads, immediately photocured in situ under UV light. Polymer concentration (100 and 150 mg/mL) and flow rate were optimized to achieve monodisperse hydrogel beads of approximately 1000 µm. Bead morphology was analyzed (SEM), and cell viability was tracked during encapsulation and storage. Fermentative activity was evaluated over eight days in malt broth, monitoring ethanol production.

Results
The platform successfully produced uniform hydrogel beads. Lower PEGDA concentrations yielded elastic matrices, resulting in post-encapsulation viability above 90%. Immobilized cells maintained over 50% viability after 15 days at 4 °C, significantly outperforming free cells. In the bioreactor, free cell treatment reached high turbidity by Day 4 (above 1300 units), whereas the 150 mg/mL hydrogel beads exhibited the lowest turbidity (below 200 units). This confirms the beads act as a physical barrier, preventing the release of daughter cells. The 150 mg/mL beads also showed the strongest correlation between glucose consumption and low turbidity (R^2=0.994, SE=1.15). Furthermore, the Relative Metabolic Efficiency Index (RMEI) was significantly higher for the 150 mg/mL beads (0.341) compared to 100 mg/mL beads (0.110) and free cells (0.027), indicating enhanced containment and more efficient metabolic performance per unit of turbidity.

Conclusions
Microfluidics is confirmed as a robust and scalable method for producing uniform hydrogel beads, highly suitable for S. cerevisiae encapsulation. The optimization using PEGDA, particularly the 150 mg/mL concentration, not only drastically improves cell viability during prolonged storage but also provides a superior system for fermentation. This highly efficient containment (RMEI=0.341) results in low product turbidity, streamlining downstream clarification processes and highlighting the strong potential of this technology for applications in the food and biotechnological industries.

Wednesday, October 29, 2025

VIII International Congress in Translational Medicine

AREA

Microbiology and Immunology

POSTER TITLE

FROM GENOMES TO VACCINES: A REVERSE VACCINOLOGY APPROACH AGAINST LEISHMANIA SPP.

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Introduction
Leishmaniasis are parasitic diseases caused by Leishmania spp., transmitted to humans through sandfly bites. Depending on the Leishmania species involved and the host's immune status, the clinical manifestations vary, from mild, self-healing skin ulcers (cutaneous leishmaniasis, CL) to the potentially fatal visceral leishmaniasis. Current treatments are expensive, highly toxic, and involve complex regimens. Considering these limitations, vaccination has emerged as a promising, cost-effective solution, however, no vaccines have been approved for humans yet, and the experimental vaccines evaluated to date have shown limited and variable efficacy. Here, we propose a reverse vaccinology approach to identify new immunogens that could protect against different forms of leishmaniasis.
Materials and methods
Nine vaccine targets were identified from annotated Leishmania proteomes, considering protein conservation, transcriptomic data, epitope predictions, and subcellular localization. These genes were cloned into the pVAX1 vector, and their expression in vitro was confirmed by immunofluorescence. Five candidates (Ag1-Ag5) were subsequently evaluated as prophylactic vaccines in a CL model. Groups of BALB/c mice received three intramuscular doses, administered every 28 days of 100 µg pVAX1 encoding one candidate or empty pVAX1 vector as control. Twenty-two days after the final dose, mice were challenged on the left hind footpad with fluorescent-tagged Leishmania amazonensis. Footpad thickness was measured weekly to monitor infection, and the parasite loads were analyzed using in vivo fluorescence imaging.
Results

The in vitro expression of all identified candidates was successfully demonstrated. The five candidates tested as prophylactic vaccine significantly reduced lesion size compared to the control ($p<0.005$), with Ag3 and Ag5 showing the best performance, 5 and 3-fold reduction, respectively. These results correlated with the parasitic load measured by in vivo fluorescence imaging.

Conclusions

These results demonstrate the potential of reverse vaccinology in the discovery of immunogens capable of conferring protection against leishmaniasis in a preclinical model of the disease.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Microbiology and Immunology

POSTER TITLE

PROPHAGE AND LYSIN DIVERSITY IN GROUP B STREPTOCOCCUS AS A BRIDGE TOWARD NOVEL ANTIMICROBIAL STRATEGIES

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Group B Streptococcus (GBS) remains a leading cause of neonatal sepsis and meningitis worldwide. The increasing emergence of antimicrobial resistance underscores the urgent need for alternative therapeutic strategies. In this context, bacteriophages and their lytic enzymes, lysins, represent a promising frontier in translational antimicrobial research, offering targeted, microbiota-sparing alternatives to conventional antibiotics.
This study characterizes the prophage diversity and lysin gene content in human-associated GBS isolates from Argentina (Arg-GBS) and Australia (Aus-GBS), from a genomic and phylogenetic perspective.
Methods
Genomes (n=283) from Australian (Aus-GBS) neonatal infection and maternal carriage isolates and Argentinian (Arg-GBS) isolates, also including urinary tract infections, were analyzed. Prophages were identified and validated using VirSorter2 and CheckV, respectively. VIRIDIC-like analysis, based on total average nucleotide identity (tANI), was performed to define prophage species (≥95% tANI), and genera (≥70% tANI). Additionally, prophage groups previously reported by Kovacec et al 2024, were considered. A Neighbor-Joining (NJ) tree was constructed to infer the phylogenetic relationships among the phages. Associations between phage species and host features (geographic origin, sequence type, or clonal complex) were assessed using the uncertainty coefficient. Gene annotation was carried out using Pharokka. Phages were annotated to identify putative lysins.
Results
We identified 360 prophages, with half of the Aus-GBS isolates carrying at least one prophage (50.9%). Phages were distributed across more than 20 clonal complexes, being particularly abundant in CC17 and CC19, both associated with invasive neonatal disease. Comparative clustering and phylogenetic analyses revealed both shared and geographically structured prophage populations, suggesting local co-evolution with host strains. Importantly, distinct lysin genes were detected across phage clusters, displaying diversity in catalytic and binding domains that may underlie differences in host specificity and antibacterial potential.
The conservation of certain lysins across continents highlights stable enzymatic scaffolds for therapeutic development, while country-specific variants reveal adaptive features with translational potential, underscoring that understanding prophage and lysin diversity in GBS is key to advancing next-generation antimicrobial and precision enzybiotic strategies. From a translational perspective, these findings provide a genomic foundation for the rational selection and engineering of GBS-specific lysins as potential enzybiotics.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Oncology

POSTER TITLE

STAT3 as a target in Breast Cancer treatment: Flubendazole repositioning versus siRNA silencing

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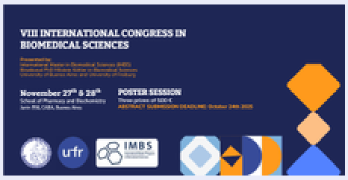
POSTER ABSTRACT (350 WORDS MAXIMUM)

Metastasis accounts for 90% of breast cancer (BC) deaths. Within BC tumors, poorly-differentiated cells, known as BC stem-like cells (BCSLCs), are considered responsible of tumor initiation and dissemination. BCSLCs are key candidates for novel therapies, such as nanoparticle-mediated drug delivery, aiming to selectively distribute therapeutics. Signal transducer and activator of transcription 3 (STAT3) is an early tumor diagnostic marker which promotes BC progression, proliferation, metastasis and chemoresistance, and is deregulated in BCSLCs. This subpopulation is also characterized by a high expression of CD44, a hyaluronic acid receptor that recognizes other extracellular matrix components, like dermatan sulfate (DS).
Our previous reports indicate the selective bioactive drug distribution by polyelectrolyte complexes (PECs)

of DS, produced by local industry, and chitosan (CT): CD44 receptor and DS mediate this interaction. Aiming to take advantage of this knowledge we developed PECs loaded with (1) Flubendazole (Flu-PECs), a benzimidazole recently reported as a STAT3 inhibitor, or (2) anti-STAT3 siRNA (siRNA-PECs), to modulate this signaling pathway via drug repositioning or gene therapy.
Flu-PECs exhibit a desirable size by Dynamic Light Scattering (397±77nm) and efficient encapsulation. Their uptake was significantly higher in MDA-MB-231 cells (invasive phenotype, 98%CD44 expression) than in MCF-7 cells (less invasive, 57%CD44 expression), and minimal in a healthy breast cell line (MCF-10A, 95%CD44 expression), evaluated by flow cytometry and confocal microscopy. Flu-PECs selectively interact with the BC CD44 through DS, confirmed in DS blocking experiments. Flu-PECs inhibited nuclear translocation of phosphorylated STAT3, reduced C-Myc and BCL-2 expression ($p<0.01$, $p<0.05$ respectively), induced G2/M arrest and apoptosis. In mammospheres (cultures enriched in BCLSCs), Flu-PECs were detected in the inner layers of the spheroid, and downregulated SOX2, a master gene of indifferentiation.
siRNA-PECs (202±28nm) also exhibit a higher uptake in MDA-mb-231 compared to MCF-7. siRNA-PECs achieved effective STAT3 silencing, correlating with a G2/M cell cycle arrest.
The nanoparticles encapsulating Flu or siRNA target the overexpressed CD44 receptor on BCLSCs, a subpopulation associated with worst prognosis. Both nanoformulations exhibit anti-cancer efficacy through STAT3 inhibitor or gene therapy. This interdisciplinary approach brings together cell biology, molecular targeting and nanotechnology, while fostering collaboration with national pharmaceutical development.



Tuesday, October 28, 2025

VIII International Congress in Translational Medicine

AREA

Oncology

POSTER TITLE

MOLECULAR PROFILING OF PEDIATRIC MEDULLOBLASTOMA IN ARGENTINA: INTEGRATING TECHNIQUES FOR PRECISION DIAGNOSIS IN RESOURCE-CONSTRAINED COUNTRIES

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background: Medulloblastoma (MB), an embryonic tumor of the cerebellum, is the most common malignant pediatric tumor of the central nervous system (CNS) and shows marked molecular and clinical heterogeneity. The 2021 WHO classification of tumors of the CNS recognizes four main molecular groups (MG): i) WNT-activated, ii) SHH-activated and TP53-wildtype (SHH TP53-), iii) SHH-activated and TP53-mutant (SHH TP53+), and iv) non-WNT/non-SHH group defined by molecular alterations, each one with distinctive prognosis.
Aim: To characterize molecular alterations in MB, assess MG and correlate them with risk, histology, location, and overall survival (OS).

Methods: Molecular alterations were detected in 84 pediatric MB cases from Argentina using an integrative workflow combining immunohistochemistry, fluorescent in-situ hybridization, Sanger sequencing and NanoString-based classification.
Results: Non-WNT/non-SHH tumors predominated (60.7%), followed by SHH (27.4%) and WNT (10.7%). The most prevalent alterations were YAP1 overexpression and TP53 gene in 24/84 (28.5%) cases each, followed by GAB1 overexpression in 19/84 (22.6%). χ^2 test of independence showed association between MG and genetic alterations (χ^2, P=6.72x10-17), particularly group-specific alterations included CTNNB1 and chromosome 6 monosomy in WNT (Standardized Residuals (SR) = |4.38| and |4.90|), and MYCN and i17q in non-WNT/non-SHH (SR=|2.85| and |2.58|). GAB1 and YAP1 overexpression were significantly associated with SHH TP53- (SR=|2.86| and |3.16|). Statistically significant association between the MG and biological risk was disclosed (χ^2, P=1.71x10-10), namely WNT were associated with low biological risk (SR=|4.54|), while SHH TP53+ showed association with very high risk (SR=|4.23|). SHH TP53- were overrepresented in the standard-risk category (SR=|2.44|). The OS differed among the MG; WNT presented a higher OS as compared to SHH TP53+ (Kaplan-Meyer, Log-rank (Mantel-Cox) Test, P=0.026) and the non-WNT/non-SHH group (P=0.038). Only the presence of TP53 alteration was associated with a decrease in OS (P=0.022).
Conclusions: In combination, lower-complexity methods resolved 90% of cases, while NanoString clarified the remaining, achieving 98.8% precision. These findings reinforced the value of molecular classification in the prognostic stratification of pediatric MB patients. This first MG characterization of pediatric MB in Argentina demonstrated that a tiered, conventional-tools-based approach can deliver high diagnostic accuracy and prognostic value, supporting its integration into routine care in resource-limited countries.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Pharmacology and Toxicology

POSTER TITLE

"VDAC as a Novel Drug Target in Trypanosoma cruzi: Evidence from Small-Molecule Inhibition and Mitochondrial Dysfunction"

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Chagas disease, caused by Trypanosoma cruzi (T. cruzi), remains a major public health issue in Latin America and worldwide. Currently available drugs show limited efficacy and significant adverse effects, underscoring the urgent need for safer and more effective therapeutic alternatives. Voltage-dependent anion channels (VDACs), located in the outer mitochondrial membrane, regulate metabolite flux and mitochondrial energy homeostasis. Free tubulin and NADH modulate VDAC conductance. Recently, small-molecule inhibitors (SMIs) such as SC18 and X1 have demonstrated anticancer potential by targeting human VDACs. SC18 binds to the internal NADH-binding pocket, while X1 antagonizes tubulin-mediated

VDAC inhibition.

Given that multiple VDAC orthologs are encoded in the T. cruzi genome and that CRISPR/Cas9-based studies have revealed their role in parasite replication and mitochondrial function, we investigated the antiparasitic activity of SC18 and X1. SC18 reduced trypomastigote viability by 75% at 10 μM, while X1 induced complete lysis at all concentrations tested. Against intracellular amastigotes, SC18 showed an IC_{50} of 7.33 μM (95% CI: 5.17–10.25), whereas X1 displayed an IC_{50} of 4.42 μM (95% CI: 2.78–6.84), but with high cytotoxicity in Vero cells (selectivity index <10). In vivo treatment with SC18 partially reduced parasitemia but did not improve survival rates.

To validate VDAC as a mitochondrial target in T. cruzi, we assessed mitochondrial membrane potential (ΔΨm) using TMRM-based flow cytometry. SC18 produced a dose-dependent decrease in TMRM fluorescence intensity, with median FITC-A values dropping from 170 (control + TMRM) to ~39 (SC18-treated), closely resembling CCCP-induced depolarization. These results confirm that SC18 disrupts mitochondrial bioenergetics through VDAC inhibition, providing functional validation of VDAC as a novel druggable target in T. cruzi.

Collectively, our findings establish VDAC as a promising therapeutic target for Chagas disease and support the rational design of optimized SMIs with enhanced selectivity and potency.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Pharmacology and Toxicology

POSTER TITLE

LAB-ON-A-CHIP AND IMAGE ANALYSIS SYSTEM TO EVALUATE FENOFIBRATE'S IMPACT ON TRYPANOSOMA CRUZI ALONE AND IN COMBINATION WITH BENZNIDAZOLE

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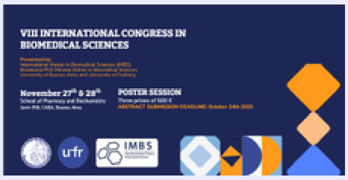
POSTER ABSTRACT (350 WORDS MAXIMUM)

Chagas disease, caused by Trypanosoma cruzi, presents a significant therapeutic challenge. The standard drug, Benznidazole (BZN), has limited effectiveness during the chronic phase and can cause severe side

effects. Coadjuvant therapies that modulate the host's inflammatory response offer a promising strategy to improve treatment. Fenofibrate (FEN), a PPAR-α ligand currently used as a hypolipidemic agent, is known to attenuate cardiovascular inflammation, but its role in T. cruzi infection remains unclear. This study aimed to evaluate the in vitro effect of Fenofibrate, both as a monotherapy and in combination with BZN, on T. cruzi infection.
First, the effect of Fenofibrate on trypomastigotes, infected cells, and its potential to enhance BZN was investigated using an innovative Lab-on-a-Chip (LOC) device. This tool automatically generates concentration gradients, enabling the simultaneous evaluation of different concentrations for both single-drug and combination therapies. Quantification was carried out using software that counts free trypomastigotes. For infected cells, this system assessed the success of the intracellular cycle by specifically counting the parasites released into the medium.
Subsequently, standard in vitro assays were conducted. Infection assays tested Fenofibrate in both pre-treatment (prophylactic) and post-treatment (therapeutic) schedules on VERO (non-phagocytic) and RAW 264.7 (macrophage) host cells. Also, viability assays were performed on both cell lines.
The results demonstrated that Fenofibrate, as a monotherapy, significantly reduced T. cruzi viability in a dose-dependent manner in both free trypomastigotes and infected cells. Subsequent combination assays revealed that Fenofibrate did not alter the trypanocidal action of BZN against either target. In VERO cells, neither pre- nor post-treatment with the drug significantly altered parasite release. In contrast, treatment of RAW cells resulted in reduced trypomastigote release compared with the control, suggesting a cell-type-dependent modulatory effect. Additionally, the drug's therapeutic window was determined.
These findings highlight the LOC´s versatility for evaluating complex drug interactions and confirm the relevance of cell type to fenofibrate´s action. Furthermore, this work supports the potential benefit of combining fenofibrate with benznidazole, highlighting its promise as a therapeutic adjunct in Chagas disease.



Thursday, October 30, 2025

VIII International Congress in Translational Medicine

AREA Pharmacology and Toxicology

POSTER TITLE Study of the interaction of Flubendazole-loaded polyelectrolyte complexes with the endothelium, for a potential in vivo administration in breast cancer treatment

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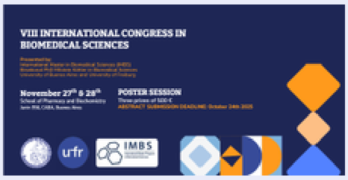
POSTER ABSTRACT (350 WORDS MAXIMUM)

Despite decades of declining mortality through earlier detection and advancements in treatment, breast cancer (BC) remains the second leading cause of cancer death among women. In this study a new nanoformulation is used to distribute flubendazole (Flu), a poor soluble anthelmintic recently repurposed as an antitumoral agent. We reported its encapsulation in Chitosan/Dermatan Sulfate polyelectrolyte complexes (Flu-PECs). This nanoformulation selectively deliver Flu to BC cell lines, decrease their invasiveness and induce apoptosis-mediated death. To improve the understanding on biodistribution of Flu-PECs, the present study aimed to analyze their effects on endothelial cells. Flu-PECs were synthesized through ionotropic gelification and characterized by Dynamic Light Scattering (DLS) and Transmission Electron Microscopy (TEM) at different pH values. Also, stability of PECs was assessed by DLS along 14 days. The endothelial cell line EAhy926 was employed for cytotoxicity studies by the MTT assay. Besides, the binding and internalization studies of fluorescently-labeled PECs were performed with or without a lipopolysaccharide (LPS) pretreatment, by fluorescence microscopy studies. Flu-PECs were synthesized at pH 3.5, and exhibit an efficient encapsulation, equivalent to 30 μM. Their hydrodynamic diameter is

353±41nm, which remains stable for 14 days. Size decreases to 217.2±12nm at endosomal pH (4.5) and 102.6±13nm at physiological pH (7.2). Cytotoxicity assays in EAhy-926 revealed reduced viability for Flu in normal conditions, while the same concentration of encapsulated Flu in PECs does not induce cytotoxicity. However, after LPS treatment both Flu and Flu-PECs decrease cell viability similarly (65±16% vs. 66±10%). Fluorescence studies show that Flu-PECs have an increased uptake in LPS-activated cells. The obtained results show that Flu-PECs have stable and desirable physicochemical properties for in vivo administration, and can selectively recognize the activated endothelium, thus preventing cytotoxicity in the healthy one. Overall, the encapsulation of this repurposed anthelmintic could prove a smart solution for its selective distribution towards BC cells and their characteristic vasculature.



Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA
Pharmacology and Toxicology

POSTER TITLE
Sex-dependent differences in glial modulation of neuronal phenotype in a synaptopathy model

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Autism spectrum disorder (ASD) is a neurodevelopmental synaptopathy characterized by behavioural deficits, altered synaptic profiles, and neuroinflammation involving cytokine upregulation and microglial activation. A consistent 4:1 male-to-female ratio and sex-related differences in ASD symptoms have been documented, yet the mechanisms underlying these dimorphisms remain poorly understood. Here, we investigated whether glial cells modulate synaptic and dendritic remodelling in a sex-dependent manner. Wistar rat pups were prenatally exposed (E10.5) to valproic acid (VPA, 450 mg/kg, i.p.) or saline (control). Cortical microglial and astroglial cells were isolated from control and VPA pups and cultured to obtain microglial- and astroglial-conditioned media (MCM and MCA). Cortical neurons were isolated from the same groups, cultured for seven days, and exposed to different MCM and MCA concentrations. Dendritic (MAP2) and synaptic (synaptophysin, SYN) profiles were analyzed by immunocytochemistry. In the absence of glial soluble factors, neurons isolated from VPA male pups showed a preserved dendritic tree and an increased number of SYN puncta compared to neurons from control animals. In contrast, neurons from VPA female pups did not differ from controls in these parameters. When exposed to MCM, neurons from control male animals showed reductions in both dendritic tree area and SYN puncta number. In neurons from VPA male animals, MCM also decreased MAP2 dendritic area, but a higher concentration was required to reduce SYN puncta number. Exposure to MCA decreased dendritic tree area in both

control and VPA male neurons, without affecting SYN puncta. In females, MCM reduced SYN puncta number without modifying dendritic morphology in neurons from control and VPA. MCA decreased SYN puncta number and dendritic tree area similarly in both groups. These findings indicate that neurons from VPA males are more resistant to glial modulation compared to those from control males, whereas neurons from VPA females do not differ from control females. Thus, glial soluble factors regulate dendritic and synaptic remodelling in a sex-dependent manner under both basal and VPA-induced conditions, revealing distinct mechanisms of vulnerability and resistance between males and females.

Wednesday, October 22, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

Brain-Heart Axis Protection Induced by Donepezil and Nebivolol in a Mouse Model of Cerebral Ischemia/Reperfusion

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Objective: Although stroke-induced cardiac injury has been studied in the last decades, differences between ischemia (I)- and reperfusion (R)-induced cardiac damage have not yet been established. It is known that cardiac electrical and contractile alterations complicate the clinical course of stroke. We aim to evaluate the differences between I and R over cardiac function, and the protective effects of donepezil (DPZ) and nebivolol (NBV) on cerebral infarct size (IS), left ventricular dysfunction, and electrocardiographic (ECG) abnormalities induced by cerebral I and R.
Methods: Male FVB mice underwent 1h of cerebral I and 24h of R. DPZ (2 mg/kg, s.c.) enhances parasympathetic tone, while NBV (10 mg/kg, i.p.) provides selective β1-blockade. Neurological and cardiac outcomes were assessed by IS, neurological score, survival, ECG, echocardiography, heart rate variability (HRV), arrhythmias, and mRNA expression by PCR (Connexin-43 (Cx-43), IL-1 β).
Results: IS was 36.4 ± 3.3% in I/R, 26.7 ± 3.8% in I/R+DPZ (p=NS vs. I/R), and 11.9 ± 2.3% in I/R+NBV

(p<0.0001 vs. I/R). NBV improved neurological score at 24hR. Survival was 43.3% in I/R, 50.0% with DPZ, and 72.7% with NBV. Both DPZ and NBV preserved ejection fraction (75.8±1.1% and 71.2±1.9%, respectively) compared with I/R (67.7±1.5%; p<0.001 and p=0.04, respectively). QTc prolonged at 1hI in I/R (143.0±4.1 ms vs. baseline 125.6±3.3; p<0.01), was prevented by DPZ (132.6±2.0 vs. 124.0±4.5; p=NS) and NBV (143.5±4.5 vs. 137.4±3.4; p=NS). At 24hR, QTc further increased in all groups (p<0.01 vs. baseline). Tp–Te also increased during R in I/R (35.2±1.6 vs. 26.9±1.2 ms; p<0.01), I/R+DPZ (47.1±3.3 vs. 32.6±3.4; p<0.0001), and I/R+NBV (49.3±3.1 vs. 33.8±2.0; p<0.01). I/R reduced total HRV and parasympathetic tone, while both DPZ and NBV preserved it throughout I/R. NBV prevented myocardial Cx-43 expression reduction, while both drugs attenuated the increase in IL-1β mRNA.
Conclusion: Cerebral I/R induces differential cardiac depolarization and repolarization abnormalities, significantly increasing ventricular repolarization and dispersion during reperfusion. NBV reduced infarct size, improved neurological outcomes, increased survival and restored Cx-43 after cerebral I/R. Both DPZ and NBV prevented systolic dysfunction, total HRV and parasympathetic reduction, attenuated depolarization/repolarization abnormalities during ischemia, although not during reperfusion, and prevented IL-1β mRNA increase.

Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA	Physiopathology
POSTER TITLE	REMOTE ISCHEMIC PRECONDITIONING ATTENUATES POST-INFARCTION VENTRICULAR REMODELING VIA A THIOREDOXIN-1–DEPENDENT MECHANISM

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Myocardial infarction (MI) remains a leading cause of mortality in industrialized countries. Remote ischemic preconditioning (RIPC), induced by brief cycles of ischemia and reperfusion in distant tissues, is a cardioprotective strategy known to reduce infarct size in acute ischemia/reperfusion models. However, its role in post-infarction ventricular remodeling remains unclear. This study aimed to evaluate the effects of RIPC on ventricular remodeling and to explore the potential role of Thioredoxin-1 (Trx-1) in this process. In a murine model of MI without reperfusion, three experimental groups were evaluated at 7 and 28 days post-infarction: (1) Sham (thoracotomy without coronary ligation), (2) MI (permanent left coronary artery

ligation), and (3) MI + RIPC (three 5-min cycles of femoral artery ischemia/reperfusion applied prior to coronary ligation). At 7 days, Wild-type (WT) and transgenic mice expressing a dominant-negative form of Trx-1 (DN) were studied. Ventricular function, infarct expansion, and molecular markers of extracellular matrix remodeling (MMP-2, MMP-9), inflammation (TNF-α, IL-6, IL-10), oxidative stress (protein carbonylation, superoxide dismutase activity), and Trx-1 expression were analyzed. At 28 days, infarct size, ventricular performance, cardiac hypertrophy, and myocardial collagen and capillary density were assessed.

At 7 days post-MI, ejection fraction and expansion index indicated preserved systolic function and reduced ventricular dilation in the RIPC group compared with MI. RIPC attenuated MMP-9 activity and pro-inflammatory cytokine expression (TNF-α, IL-6) while enhancing IL-10. Oxidative stress was mitigated, as evidenced by lower nitrate/nitrite, nitrotyrosine, protein carbonyls, and 4-HNE adducts. Trx-1 expression was upregulated in RIPC, whereas SOD activity decreased, suggesting improved redox balance. In DN mice, the protective effects of RIPC on cardiac function were abolished. However cytokine modulation, and oxidative stress remained improved as seen in WT mice. At 28 days, RIPC preserved left ventricular function, reduced interstitial collagen deposition in the remote zone, and increased capillary density, indicating sustained antifibrotic and proangiogenic effects independent of cardiac hypertrophy.

RIPC confers both early and long-term protection against post-infarction ventricular remodeling through anti-inflammatory, antioxidant, and antifibrotic mechanisms. These effects depend on functional Trx-1, identifying this redox protein as a key mediator of RIPC-induced cardioprotection and a potential therapeutic target in ischemic heart disease.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

THE IMBALANCE OF THE RENIN-ANGIOTENSIN SYSTEM IN SKELETAL MUSCLE OF MALE AND FEMALE RATS INDUCED BY A HIGH-FAT DIET

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POSTER ABSTRACT (350 WORDS MAXIMUM)

The pressor axis of the renin-angiotensin system (RAS) influences mitochondrial function and promotes pro-inflammatory and pro-fibrotic states in skeletal muscle (SM), thus contributing to muscle wasting and sarcopenia. However, the evidence is limited regarding the role of obesity in intramuscular RAS and whether sex differences exist.
Objective: To evaluate the expression of RAS components in SM in male (m) and female (f) rats fed a high-fat diet (HFD) and whether RAS of obese rats is influenced by sex.
Methodology: m and f Wistar rats were fed either a HFD, or a Standard Diet (SD) from weaning until 14-week-old. Following the experimental period, body weight (BW) and systolic blood pressure (SBP) were measured. Fasting glycemia and insulinemia were determined in blood samples. The animals were euthanized, and SM was removed and processed to evaluate angiotensin (ANG) II and ANG-(1-7) levels by RIA, and AT1 and AT2 receptors by immunohistochemistry. Protocol approved by CICUAL-FFyB. Results are expressed as mean±SEM. Statistical analysis was performed using two-way ANOVA followed by Bonferroni's post-hoc test. n=10/group. *p<0.05; **p<0.01; ***p<0.001 vs. mSD; ##p<0.01; ###p<0.001 vs. fSD; &p<0.05; &&&p<0.001 vs. mHFD; diet-sex interaction: $$p<0.01.
Results: mHFD and fHFD showed higher BW (mSD=492±10, mHFD=553±8***, fSD=348±6***, fHFD=429±11###&&& g), fasting glycemia (mSD=122±6, mHFD=135±3**, fSD=117±1, fHFD=131±4## mg/dL) and insulinemia (mSD=1.1±0.1, mHFD=1.6±0.1*, fSD=0.9±0.1, fHFD=1.6±0.2## ng/mL) compared to SD groups, without differences in SBP (mSD=127±3, mHFD=132±1, fSD=127±3, fHFD=129±3 mmHg). There were no differences shown for SM/tibia length ratio (mSD=49.9±1.4, mHFD=50.1±0.8, fSD=38.1±1.2***, fHFD=40.8±1.2&&& g/cm) between diets. Regarding the RAS, ANGII (mSD=11.5±2.1, mHFD=17.4±1.2*, fSD=14.6±1.0, fHFD=11.6±0.8&$$ pg/100mg tissue) and AT1R (mSD=3.7±0.7, mHFD=7.4±0.7**, fSD=6.0±0.7*, fHFD=5.9±0.6$$ %) were increased and AT2R (mSD=7.4±1.2, mHFD=4.0±0.7*, fSD=4.3±0.8*, fHFD=3.5±0.5 %) was diminished in mHFD compared to mSD. Conversely, f rats showed no differences in RAS components between diets, while fHFD rats had lower concentrations of ANGII than mHFD. ANG(1-7) showed no differences between groups (mSD=20.0±0.5, mHFD=19.9±0.4, fSD=19.6±0.7, fHFD=20.1±0.5 pg/100 mg tissue).
Conclusion: SM from mHFD rats showed changes in RAS that could represent greater ANGII bioavailability in the tissue and its blood vessels, contributing to muscle function impairment. SM from f rats did not show modifications in RAS, suggesting certain protection in obesity.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

Absence of CB1 Receptor Impairs Motor Learning and Reduces Synaptic Vesicle Density in Mice

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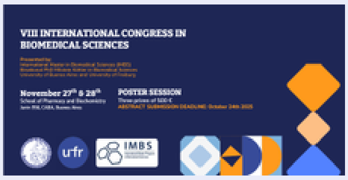
POSTER ABSTRACT (350 WORDS MAXIMUM)

The cannabinoid receptor type 1 (CB1R) is widely expressed throughout the central nervous system, in both neurons and glial cells. It plays a pivotal role in multiple physiological processes, including memory, learning, motor coordination, anxiety regulation, and mood control. Moreover, CB1R modulates synaptic transmission and neuronal plasticity, both fundamental for the adaptive responses of the nervous system. Previous studies from our group showed that CB1R-deficient mice ($CB1R^{-}/^{-}$) display reduced dendritic and axonal arborization, along with alterations in synaptic organization.

Objective and Methodology: This study aimed to evaluate the impact of CB1R absence on neuronal morphology and synaptic connectivity in the striatum and motor cortex. Sixty-day-old $CB1R^{+}/^{+}$ (wild-type) and $CB1R^{-}/^{-}$ (knockout) mice were used. Motor coordination and balance were assessed through the rotarod test over ten consecutive sessions. Expression levels of CB2R and GPR55 were quantified by RT-PCR. Coronal brain sections were processed for immunohistochemistry using primary antibodies against Synaptophysin, MAP2, and NF200. The relative area occupied by $MAP2^{+}$ and $NF200^{+}$ fibers was quantified at 20× magnification, and Synaptophysin immunoreactivity was analyzed through relative optical density (ROD).

Results: No significant differences were found in the cortical or striatal expression of CB2R (p=0.14) or GPR55 (p=0.3) between groups. In the rotarod test, $CB1R^{+}/^{+}$ mice exhibited a progressive improvement in motor performance starting from trial 4, indicative of motor learning and task adaptation. In contrast, $CB1R^{-}/^{-}$ mice failed to improve across sessions, suggesting deficits in motor learning or coordination. Dendritic arborization (MAP2, p=0.37) and axonal structure (NF200, matrix p=0.91; striosomes p=0.34; motor cortex p>0.05) did not differ significantly between genotypes. However, a significant reduction in synaptic vesicle density was observed in $CB1R^{-}/^{-}$ mice compared to controls (p=0.0039).

Conclusion: Our results indicate that CB1R deletion does not significantly alter CB2R or GPR55 expression, although a compensatory mechanism cannot be excluded. The absence of CB1R leads to impaired motor learning and a marked decrease in synaptic vesicle density, highlighting the essential role of CB1R in maintaining synaptic integrity and neuronal plasticity necessary for proper motor function.



Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

ISCHEMIC POSTCONDITIONING: DO METABOLIC CONDITIONS HAVE AN IMPACT ON ITS EFFECTS IN HEART SUBJECTED TO ISCHEMIA-REPERFUSION?

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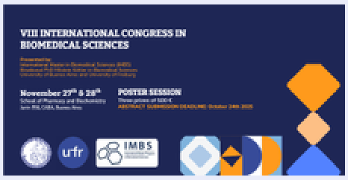
POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and aims: Ischemic postconditioning (IPostC), a cardioprotective strategy which consists of brief cycles of reperfusion (R)- ischemia (I) after the ischemic period, has elicited interest for its potential

translation to the clinical practice. However, this translation has not been successfully achieved yet. In this study, we aimed to investigate the effects of different metabolic conditions in IPostC-mediated cardioprotection.
Methods: Hearts from female Sprague-Dawley rats (220 - 270 g), previously fed ad libitum or fasted for 24 h, were isolated, perfused using the Langendorff method, and stabilized for 25 min. Hearts were then subjected to 25 min I - 60 min R. IPostC consisted of 6 cycles of 10 sec R - 10 sec I at the onset of R. Contractile function (Rate-pressure product, peak contraction rate, left ventricular end diastolic pressure and peak relaxation rate) was assessed using a latex balloon and pressure transductors during the experiments. Infarct size was determined by the TTC method. Tissue ATP content was assessed using the luciferin/luciferase bioluminescence assay. Mitochondrial ultrastructure was evaluated by transmission electron microscopy and mitochondrial number and area were analyzed using the Image J software. ATP synthesis rate, mitochondrial membrane potential ($\Delta\Psi m$) and respiratory complexes I-III, II-III and IV activities were evaluated in isolated mitochondria. ANOVA, n=6/group.
Results: In hearts from fed rats, IPostC improved contractile function (both systolic and diastolic), decreased infarct size and increased post-ischemic tissue ATP content ($p<0,05$ vs. fed Control). In hearts from fasted rats, however, there were no significant differences in these parameters between the IPostC and Control groups.
In hearts from fed rats, IPostC preserved the ultrastructure, number and size of mitochondria, and improved mitochondrial ATP synthesis rate and Complex I-III and II-III activity ($p<0,05$ vs. fed Control). These beneficial effects of IPostC were not observed in hearts from fasted rats. $\Delta\Psi m$ was also preserved by IPostC in fed rat hearts, while IPostC hearts from fasted rats showed greater despolarization ($p<0,01$ vs. IPostC fed).
Conclusion: These results suggest that fasting abolishes the beneficial effects of IPostC. This underlines the importance of further investigation on the influence of cellular metabolism in cardioprotective strategies.



Thursday, October 30, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology and General Medicine

POSTER TITLE

Integrating Genetic and Biochemical Markers to Differentiate Familial from Multifactorial Chylomicronemia in Severe Hypertriglyceridemia

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims: Familial chylomicronemia syndrome (FCS) is a rare autosomal recessive disorder, characterized by severe hypertriglyceridemia (sHTG), caused by mutations in five main canonical genes (LPL, APOA5, APOC2, GPIHBP1 and LMF1); its genotype distribution in our populations is still scarcely reported. Given the difficulty of genetic testing, and the fact that many clinically consistent cases yield negative results, several scores including lipoprotein indexes have been proposed to predict FCS. This study aimed to analyze the genotype distribution, genotype-phenotype correlation, and the predictive value of lipoprotein indexes and LPL activity in a Latin American sHTG cohort.
Methods: Serum lipid profile was performed in 61 sHTG subjects (TG>880 mg/dl), 27 genetically diagnosed as FCS and 34 with multifactorial chylomicronemic syndrome (MCS). Triglyceride/total-cholesterol (TG/TC), TG/ApoB indexes were calculated; LPL activity was measured in post-heparin plasma using a radiometric assay. A Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the discriminatory power of selected variables.
Results: In FCS patients, LPL activity was below 25% of normotriglyceridemic individuals. Variants in LPL and APOA5 showed similar prevalence (36% and 36% respectively) followed by GPIHBP1(12%), APOC2 (8%), and LMF1 (8%). ROC curve analysis showed excellent discrimination for LPL activity (AUC 0.972;

95% CI 0.893–0.997; p<0.0001). The optimal cut-off was ≤23.65% (Youden's J=0.971), yielding 100.0% sensitivity (95% CI 87.2–100.0) and 97.1% specificity (95% CI 84.7–99.9). TG/TC and TG/ApoB also performed well (AUC 0.844; 95% CI 0.731–0.923; p<0.0001, and AUC 0.842; 95% CI 0.728–0.922; p<0.0001, respectively). The corresponding Youden-based thresholds were >3.83 for TG/TC (sensitivity 82.8%, specificity 76.5%) and >10.96 for TG/ApoB (sensitivity 72.4%, specificity 88.2%).
Conclusion: The genotypic distribution observed in this cohort differs from that previously reported in other regions. Since no differences were found in the lipid profile according to genotype, the inclusion of complementary biomarkers in proposed algorithms is required to improve diagnostic accuracy. The use of LPL activity together with simple indexes with predictive capacity provides an accessible and applicable diagnostic tool, particularly valuable in low-resource settings.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

Evaluation of LDL-C Estimation Methods Using the Lipid Ratio Plot as a Surrogate for Beta-Quantification in a Large Argentine Population.

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims
Low-density lipoprotein cholesterol (LDL-c) remains the primary therapeutic target for cardiovascular disease (CVD) prevention. However, the gold standard beta-quantification (BQ) method is impractical for routine use. Several equations, including Friedewald, Martin-Hopkins, and Sampson, are commonly applied to estimate LDL-c, but their accuracy varies, especially in patients with hypertriglyceridemia. To overcome the lack of BQ availability in most laboratories, the Lipid Ratio Plot (LRP) has been proposed as an indirect graphical approach to assess LDL-c measurements. This study aimed to evaluate the performance of

direct LDL-c (D-LDL-c) and calculated LDL-c equations using the LRP as a surrogate for BQ in a large cohort from Argentina.
Methods
A retrospective analysis was performed on 22,748 lipid profiles from patients at Hospital de Clínicas “José de San Martín” (Buenos Aires, Argentina) between 2018 and 2022. LDL-c was measured directly by a homogeneous enzymatic assay and estimated using the Friedewald (F-LDL-c), extended Martin-Hopkins (ME-LDL-c), and Sampson (S-LDL-c) equations. Participants were classified according to triglyceride levels and LDL-c treatment goals. The LRP was used to compare each method’s regression line with the expected BQ line, evaluating concordance in the overall and hypertriglyceridemic (HTG) populations. The study was approved by the institutional ethics committee.
Results
In the total population, S-LDL-c (Y = −36.6x + 120) showed the best alignment with the BQ regression line (Y = −34.2x + 115), indicating superior accuracy. D-LDL-c also performed well (Y = −40.3x + 128), while F-LDL-c displayed the highest negative bias (Y = −49.8x + 130) and ME-LDL-c tended to overestimate LDL-c (Y = −26.7x + 108). In the HTG subgroup, S-LDL-c (Y = −38.6x + 117) and D-LDL-c (Y = −39.4x + 126) maintained high concordance with BQ, whereas F-LDL-c (Y = −68.9x + 258) and ME-LDL-c (Y = −25.0x + 104) showed greater deviation.
Conclusions
Using the LRP as a graphical surrogate for BQ, S-LDL-c and D-LDL-c demonstrated the most accurate LDL-c estimations, particularly in hypertriglyceridemic patients. The LRP proved to be a practical, innovative tool for indirectly assessing the accuracy of LDL-c methodologies and validating new estimation formulas in the absence of BQ.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA	Physiopathology and General Medicine
POSTER TITLE	EXERCISE AND TRX-1 OVEREXPRESSION: SYNERGISTIC EFFECT TO MITIGATE ISCHEMIA/REPERFUSION INJURY IN MIDDLE-AGED MICE.
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THE POSTER APPLIES FOR PRIZE	NO
Terms and Conditions	Accepted

POSTER ABSTRACT (350 WORDS MAXIMUM)

Previously, we demonstrated that voluntary wheel running and thioredoxin-1 (Trx1) overexpression independently reduce infarct size after acute ischemia/reperfusion (I/R) in young mice; however, in middle-aged mice, Trx1 cardioprotection was abolished. This study investigated whether voluntary exercise restores Trx1 cardioprotection in middle-aged mice. Hearts from wild-type (Wt) and transgenic mice overexpressing Trx1 or a dominant negative mutant of Trx1 (DN) were used. They were divided into exercise (E) or sedentary (S) groups and housed in cages with running wheels for 8 weeks. Afterwards, hearts were subjected to 30min of I and 120min of R (Langendorff technique). We extended our previous analysis by including functional and morphological evaluations through electrocardiography and echocardiography. In addition, the cross-sectional area (CSA) of myocytes was assessed using the Periodic Acid–Schiff (PAS) staining method. Data are expressed as mean±SEM and $p<0.05$ was considered statistically significant. n=5 per group. Training was confirmed by greater heart rate variation after propranolol (Wt-E: -15.76±1.2 vs Wt-S: -10.19±2.5 $p<0.05$) and by an increase in CSA of myocytes (Wt-E: 4740±21 vs Wt-S: 4422±52; Trx1-E: 4883±52 vs Trx1-S: 4504±55; DN-E: 4939±107 vs DN-S: 4404±110, $p<0.05$). Exercise reduced infarct size in Trx1 (26.8±2.8, $p<0.05$) but not in Wt or DN (Wt-E: 51.2±3.6; DN-E: 68.6±5.7). No differences were observed in ejection fraction (Wt-E: 79.8±1.7 vs Wt-S: 76±5.5; Trx-E: 78.2±6.58 vs Trx-S: 79.9±0.4; DN-E: 75.4±2.5 vs DN-S: 77.3±2.6), fractional shortening (Wt-E 41.4±1.7 vs Wt-S 41.4±0.4; Trx1-E 39.2±3.9 vs Trx1-S 37.8±2.1; DN-E 40.5±6.2 vs DN-S 39.2±2.5) or isovolumetric relaxation time (Wt-E 21.8±1.7 vs Wt-S: 20.7±0.3; Trx1-E: 21.7±0.9 vs Trx1-S: 19.7±0.47; DN-E: 19.7±1.5 vs DN-S 20.7±1.4), indicating preserved ventricular function. In conclusion, 8 weeks of voluntary exercise induce a physiological hypertrophic adaptation and restores Trx1 cardioprotection by reducing infarct size in middle-aged mice overexpressing Trx1 subjected to I/R injury.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

Post-transcriptional modulation and regional ASIC1 distribution in LPS-induced inflammatory pain

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims: Inflammatory pain models are valuable for dissecting the mechanisms linking inflammation and nociception. Previous studies showed that subplantar LPS injection upregulates ASIC1 protein levels and activates ERK signaling. Here, we investigated whether this regulation involves changes in ASIC1a mRNA expression or post-transcriptional modulation by miR-485-5p, and analyzed ASIC1 distribution in peripheral sensory tissues.
Methods: Adult mice received subplantar injections of LPS or PBS. ASIC1a and miR-485-5p expression were quantified in paw tissue and dorsal root ganglia (DRG) by RT-qPCR. Immunohistochemistry was performed to evaluate ASIC1 protein distribution in DRG and plantar skin.
Results: LPS treatment significantly increased miR-485-5p levels, whereas ASIC1a mRNA remained unchanged. Immunohistochemistry revealed enhanced ASIC1 immunoreactivity in both DRG and paw skin.
Conclusions: The findings on miR-485-5p upregulation, supports a post-transcriptional mechanism regulating ASIC1 during inflammation. These findings extend our previous observations on ERK-dependent modulation, revealing spatial changes in ASIC1 expression across sensory compartments that

may contribute to peripheral sensitization in inflammatory pain.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

CURCUMIN, A NATURAL COMPOUND WITH POTENTIAL BENEFITS IN TREATING PERIPHERAL NEUROPATHIES

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Peripheral neuropathies are highly prevalent disorders, with a 2-3% prevalence in general population and up to 24% in adults over 55 years old; clinical progression is often unfavorable due to the lack of effective therapies that comprehensively address patient needs. Previous studies of our lab have demonstrated the therapeutic potential of multipotent stem cells of bone marrow or adipose tissue origin for peripheral nerve regeneration both in terms of morphology and reduction of associated neuropathic pain. Bibliography data have demonstrated the anti-inflammatory and antioxidant properties of curcumin, as well as its capacity to enhance BDNF synthesis which can promote neuroprotection and regeneration. In this context the aim of the present study is to evaluate curcumin ability to promote neuroregeneration in combination or not with bone marrow mononuclear cells transplant. For this purpose, and considering the low solubility of curcumin in biological fluids, curcumin-PLGA nanocapsules were synthetized and characterized through DLS, RMN and IR. On the other hand, adult multipotent stem cells from bone marrow or adipose tissue were incubated with PLGA nanocapsules containing different curcumin concentrations, to evaluate cell viability through MTT assay and through fluorescence microscopy with vital dyes. To evaluate the potential of curcumin-PLGA nanocapsule in neuroregeneration, the scratch assay in dorsal root ganglia cells were performed. The results obtained demonstrate that encapsulation of 6 mg/ml curcumin in chloroform led to the formation of non-homogenous PLGA nanocapsules in terms of size, which promoted a cytotoxic effect on multipotent cells. Reduction of curcumin concentration to 1 mg/ml led to more homogeneous nanocapsules but still cytotoxic for multipotent cells. Finally, a concentration of 0.25 mg/ml resulted in increased survival of CMTA cells. Preliminary results using this curcumin-PLGA nanocapsule concentration apparently accelerated scratch closure. Finding the correct concentration of curcumin may provide basis for new therapeutic strategies for treatment of peripheral neuropathies taking advantage of the anti-inflammatory and antioxidant effect of this natural compound.

Thursday, October 30, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

MITOCHONDRIAL DYSFUNCTION AND ADAPTIVE RESPONSES IN THE PANCREAS: INSIGHTS INTO ENDOTOXEMIA-INDUCED BIOENERGETIC FAILURE

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Despite significant advances in biomedical research, the molecular mechanisms underlying sepsis and endotoxemia remain not fully understood due to their complex and multifactorial nature. Pancreas is one of the organs affected during sepsis and endotoxemia, making it a potentially critical factor in the progression of systemic disease. Given the inflammatory nature of this pathology, pancreatic mitochondria could be affected, compromising tissue bioenergetics. Our aim was to evaluate mitochondrial function and dynamics in pancreas during endotoxemia.
The experimental models used were: a) In vivo model: female Sprague Dawley rats (45 days old) treated i.p. with vehicle (control group) or LPS 8 mg/kg (LPS group). b) In vitro model: AR42J pancreatic acinar cells treated with 2.5% v/v serum from the in vivo model (control or LPS). Mitochondrial function (ATP production, membrane potential) and oxidative stress markers (H_2O_2, nitric oxide) were analyzed. Additionally, the expression of mitochondrial quality control proteins (p62, LC3, DRP1, OPA1, TOMM20) was evaluated.
Mitochondrial membrane potential was observed to decrease by approximately 30% in LPS groups in vivo model (control value: 147 ± 20 mV) and the in vitro model at 24 h. Furthermore, mitochondrial ATP production was observed decreased in both the in vivo and in vitro models in LPS groups at 6 h (control values: 84.300 ± 4.824 and 3.3 ± 0.3 nmol/min mg protein respectively); Interestingly, a recovery was only observed at 24 h in vitro model. Furthermore, H2O2 production in vitro model increased as early as 2 h (control value: 0.030±0.001 nmoles/min mg of protein). Finally, in in vivo model, decreased PGC1 alpha expression and altered PINK1 and LC3 protein expression were observed in the LPS group 8 compared to the control group. Furthermore, structures consistent with mitophagy (assessed by TEM) were observed in both endotoxemia groups. Our results suggest that endotoxemia presents early mitochondrial dysfunction in pancreas. In this context, mechanisms related to mitochondrial homeostasis appear to be activated in order to restore cellular bioenergetics. The mitochondrial mechanisms activated in the pancreas during endotoxemia could be of relevance for the development of new therapeutic strategies.
* both authors equally contributed to this work.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

PHARMACOLOGICAL AND CELLULAR MODULATION OF PPARΓ IN A MODEL OF SCIATIC NERVE WALLERIAN DEGENERATION

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims
Peripheral neuropathies are disorders characterized by persistent inflammation, functional loss, and pain. Current therapies relieve symptoms but do not address the underlying mechanisms. In experimental

models of Wallerian degeneration induced by sciatic nerve crush, our group showed that bone marrow mononuclear cells (BMMCs) migrate to the injury site and exert immunomodulatory effects, reducing inflammation and neuropathic pain (Setton-Avruj et al., 2007; Usach et al., 2011; Usach et al., 2017; Piñero et al., 2023). Indomethacin, a cyclooxygenase (COX) inhibitor, decreases prostaglandin E_2 (PGE_2) and increases prostaglandin J_2 (PGJ_2), an endogenous ligand of the peroxisome proliferator-activated receptor gamma (PPARγ), suggesting an anti-inflammatory role of this pathway in injured nerves. Based on these findings, this study aimed to evaluate PPARγ modulation induced by pharmacological and cellular treatments both in vivo (sciatic nerve crush) and in vitro (primary dorsal root ganglia, DRG).
Methods
Primary DRG cultures were treated with rosiglitazone (5 or 10 µM), with or without mechanical injury, to evaluate molecular changes in the PPARγ pathway by immunofluorescence (IF). In parallel, animals underwent sciatic nerve crush and were treated with rosiglitazone (5 mg/kg, intraperitoneally), BMMCs (1×10^9 cells, systemically), or both. Additional groups included crush (untreated), sham (nerve exposure only), and control (naïve) animals. Tissues were collected at different post-treatment time points, and ipsilateral and contralateral nerves were processed for IF and western blot (WB). Poly(lactic-co-glycolic acid) (PLGA) nanocapsules containing rosiglitazone were developed to achieve targeted and sustained drug release at the injury site. Their physicochemical characterization is ongoing.
Results
In vitro, rosiglitazone-treated DRG cultures, with or without injury, showed molecular changes associated with PPARγ modulation. In vivo, nerve injury reduced PPARγ signal, while BMMC treatment induced a sustained increase, greater than that observed with rosiglitazone alone. The combined treatment promoted a more uniform activation of the pathway in the ipsilateral nerve.
Conclusions
Nerve injury alters PPARγ expression, while pharmacological and cellular treatments modulate its activation. PLGA nanocapsules loaded with rosiglitazone could provide targeted and sustained in vivo delivery. Further studies are required to confirm the potential pro-regenerative effects of rosiglitazone following nerve injury.

Sunday, October 26, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

POTENTIAL FUNCTIONAL FOOD: OPUNTIA FICUS-INDICA MODULATES COX2 IN RENAL EPITHELIAL CELLS AND PREVENTS OXALATE-INDUCED DAMAGE

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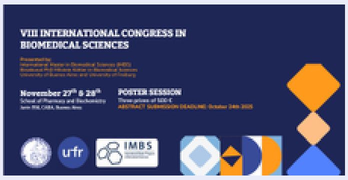
POSTER ABSTRACT (350 WORDS MAXIMUM)

Renal lithiasis is the most common disease of the urinary tract, with a global prevalence of approximately 12% and a high recurrence rate. Over 60% of patients with urolithiasis present calcium oxalate (CaOx)

stones. Oxalate nephropathy is characterized by tubulointerstitial CaOx crystal deposits, inflammation, fibrosis, and progressive renal insufficiency, potentially leading to Chronic Kidney Disease (CKD). Our lab previously demonstrated that oxalate (Ox) treatment reduces renal epithelial cell number and induces epithelial–mesenchymal transition (EMT) after 24 hours. We also demonstrated that cyclooxygenase-2 (COX2) is a cytoprotective gene whose expression and activity are essential for renal protection against hyperosmolar stress and renal epithelial cell monolayer restitution after 72 h of Ox treatment. Functional foods (FF), including natural or enriched foods, offer health benefits and may help prevent diseases like obesity, diabetes, cancer, and hypertension. In South America, Opuntia ficus-indica (Ofi), or 'nopal', is the most widely distributed cactus, especially in Northwest Argentina (NOA). Due to its availability and low cost, Ofi has potential as a food ingredient. In addition, its chemical composition, enriched in polyphenols and flavonoids, gives Ofi a high potential as an antioxidant compound. Considering that Ox-induced renal damage is initiated by oxidative stress, we evaluated whether Ofi extracts prevent Ox-induced injury and the role of COX2 in this effect. Madin-Darby canine kidney (MDCK) epithelial cells were cultured in a hyperosmolar environment (512 mOsm/Kg H_2O) for 72 hours to form a differentiated monolayer, then exposed to 1.5 mM Ox with or without 12.5 or 25 µg GAE/g of Ofi extract (from cladode flour or mucilage). Extracts were added 30 minutes before Ox exposure. After 24 hours, cell number and viability were assessed via Trypan Blue exclusion, morphology via FITC-phalloidin staining, and COX2 expression via western blot. Pretreatment with Ofi extracts preserved epithelial morphology and modulated COX2 expression, especially with mucilage extract. These findings suggest Ofi may protect renal epithelial cells from Ox-induced damage.



Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA	Cellular & Molecular Biology
POSTER TITLE	Neuroinflammatory Activation of RIPK1 through ASIC1a Signaling in Neurons
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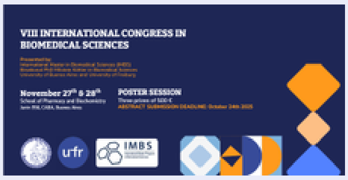
THE POSTER APPLIES FOR PRIZE	NO
Terms and Conditions	Accepted

POSTER ABSTRACT (350 WORDS MAXIMUM)

Background: Inflammation in the brain is often accompanied by extracellular acidosis and elevated levels of interleukin-6 (IL-6), both of which can alter neuronal function. In our previous work, we showed that IL-6 combined with mild acidosis (pH 6.5) promotes the translocation of ASIC1a channels to the plasma membrane, activating ERK-dependent signaling. Recent studies have suggested that ASIC1a can also recruit receptor-interacting protein kinase 1 (RIPK1) through non-conductive mechanisms. Here, we investigated whether the IL-6/pH-mediated pathway previously described could also engage RIPK1 activation in neurons.
Methods: We examined the effects of IL-6 and extracellular acidification on ASIC1a localization and signaling in heterologous HEK293 cells and primary cortical neurons. ASIC1a activity was pharmacologically modulated with MitTx, a selective activator, and PcTx-1, an inhibitor that stabilizes the channel's closed state. To mimic inflammatory conditions, cortical neurons—endogenously expressing RIPK1—were exposed to IL-6 and low extracellular pH. Phosphorylated RIPK1 (pRIPK1) was detected by immunocytochemistry as an indicator of kinase activation, while ASIC1a redistribution and downstream phosphorylation events were analyzed by Western blotting and confocal microscopy.
Results: Under control conditions, neurons displayed weak, diffuse pRIPK1 staining. Exposure to IL-6 and acidic pH significantly increased pRIPK1 signal intensity, which became concentrated in the soma and proximal processes. Pretreatment with PcTx-1 completely prevented this increase, maintaining a pattern like control cells.
Conclusion: These findings demonstrate that neuroinflammatory conditions activate RIPK1 in neurons through ASIC1a-dependent mechanisms. This supports a non-conductive signaling mode of ASIC1a and identifies a potential ASIC1a–RIPK1 axis linking inflammatory stress to neuronal injury. Unlike the transient activation mediated by ion conduction, this signaling mode may allow ASIC1a to remain active over longer periods, sustaining downstream pathways. Future studies using complementary approaches will be required to further confirm and characterize this signaling pathway



Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

MOLECULAR MECHANISMS ASSOCIATED WITH PGE2 RECEPTORS ACTIVATION IN THE RESTITUTION OF AN OXALATE-DAMAGED RENAL EPITHELIUM

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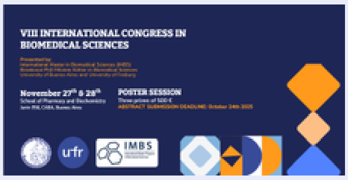
POSTER ABSTRACT (350 WORDS MAXIMUM)

Kidney stone disease is the most frequent urinary tract disorder, with a global prevalence of 12%. Calcium oxalate (CaOx), the main component of kidney stones, injures renal cells and tubular structures, contributing to chronic kidney disease. Previous results showed that renal differentiated cells treated with oxalate (Ox) for 24h exhibited a spindle-shaped morphology characteristic of epithelial–mesenchymal transition. After 48h, cells began to recover their shape, and by 72h the epithelium was almost restituted. We previously demonstrated that PGE2 is essential for epithelial restitution and that COX-2 inhibition

impedes this process. EP2 and EP4 receptor expression was confirmed by qPCR.
This study aimed to evaluate the molecular mechanisms associated with PGE2 receptors activation during restitution of Ox-injured renal epithelial cells. MDCK cells were differentiated in a hyperosmolar environment (512 mOsm/kg H2O, 72h) and then treated with 1.5 mM Ox for up to 72h. Cells were exposed to 10 µM PF-04418948 and/or 10 µM L-161,982 (EP2 and EP4 antagonists, respectively). Cell number decreased 24h after treatment with EP2a or EP4a + Ox compared to Ox alone, and at 48h a reduction was also observed with both antagonists.
Morphological analysis showed that, after 24h, antagonist-treated cells resembled Ox-treated ones, but at 48h Ox cells recovered morphology, while EP2a-treated cells showed partial restitution and EP4a or combined treatments caused greater damage. E-cadherin localization was altered by EP2a/EP4a: after 24h, internalization increased, and at 48h membrane redistribution was inhibited.
To assess EP2/EP4 activation, intracellular cAMP was measured. cAMP increased after 11h of Ox treatment but was inhibited by both antagonists. In stably transfected EP2-AmCyan1 or EP4-mTurq2 cells, both receptors showed peripheral localization, which shifted to internalization after 12h, with recovery by 72h. Confocal microscopy revealed colocalization with EEA, confirming trafficking to early endosomes.
These findings provide insight into the mechanisms triggered by EP2 and EP4 after Ox injury, showing that their activation increases cAMP, regulates E-cadherin relocalization, and promotes receptor internalization followed by recovery during epithelial restitution.



Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Cellular & Molecular Biology

POSTER TITLE

MECHANISTIC EVALUATION OF IMT504-INDUCED REMYELINATION: EFFECTS ON MICROGLIA ACTIVATION AND OLIGODENDROCYTE DIFFERENTIATION

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Demyelination involves the loss of myelin sheaths surrounding axons, while remyelination is the regenerative process that restores myelin integrity and neuronal function. Multiple sclerosis (MS) is a common inflammatory demyelinating disease in which endogenous remyelination frequently fails. IMT504 is a synthetic non-CpG oligodeoxynucleotide (ODN) of 24 nucleotides containing two conserved PyNTTTTGT motifs. Its safety has been evaluated in pharmacokinetic and toxicological studies in rats and monkeys, and a Phase I clinical trial has been recently authorized (Resol. DI-2023-6969-APN-ANMAT#MS). Considering the immunomodulatory and regenerative properties of IMT504, and previous findings by our group demonstrating its promyelinating effects in a

neuroinflammatory demyelination animal model, this study aimed to determine whether IMT504 directly affects oligodendroglial progenitor cells (OPCs) and microglia, the cell types mainly involved in remyelination.
Using in vitro assays in primary cultures, we investigated (i) IMT504 incorporation into microglia, its effects on microglial phenotypes, and the signaling pathways involved; and (ii) IMT504 incorporation into OPCs and mature oligodendrocytes (OLs), its effects on OPC differentiation, the expression of PDGFRα and MBP genes, and the signaling pathways involved. Primary cultures were obtained from cerebral cortices of 1–2-day-old rats. Microglia were treated with IMT504-Alexa488 for 16 h for incorporation assays. Also, microglia were treated with IMT504 or saline solution for 8, 16 and 24 min to analyze signaling pathway activation by Western blot, and for 24 h for qPCR analyses of pro- and anti-inflammatory cytokine gene expression. OPCs and OLs were similarly treated to evaluate IMT504 incorporation, signaling pathway activation, and PDGFRα/MBP mRNA expression.
Fluorescent IMT504 was incorporated by microglia, OPCs, and OLs. In microglia, IMT504 activated ERK1/2, p38, JNK, and NF-κB signaling, increased TNFα and IL-10 expression, and reduced IFNγ and IFNβ mRNA levels. In oligodendroglial cells, IMT504 reduced PDGFRα and increased MBP mRNA expression, consistent with enhanced OPC maturation after repeated stimulation. IMT504 also induced ERK1/2 and p38 signaling pathway activation.
Overall, these findings indicate that IMT504 modulates microglial and oligodendroglial responses through multiple signaling pathways, promoting predominantly an anti-inflammatory microglial phenotype and oligodendroglial maturation. Thus, IMT504 emerges as a promising therapeutic candidate for demyelinating diseases such as MS.

Thursday, October 30, 2025

VIII International Congress in Translational Medicine

AREA

Cellular and Molecular Biology

POSTER TITLE

LIRAGLUTIDE MODULATES PPARg, GLP-1R, AND ADIPOKINE EXPRESSION IN EXPANDED VISCERAL ADIPOSE TISSUE

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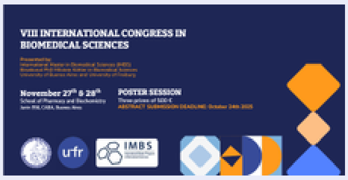
POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and aim: Central obesity is characterized by the expansion of visceral adipose tissue (VAT),

which is considered one of the main risk factors for metabolic diseases. Recently, new pharmacological approaches have been investigated for obesity treatment. We previously demonstrated that Liraglutide (LGT), a glucagon-like peptide-1 receptor (GLP-1R) agonist, reduces visceral adiposity, modulates gelatinase activity and improves vascularization and fibrosis in VAT from a diet-induced obesity model. However, several mechanisms underlying LGT effects on VAT remain unclear. Our aim was to evaluate the effect of LGT on peroxisome proliferator-activated receptor gamma (PPARɣ), GLP-1R and adipokines expression in expanded VAT from mice fed a high fat-diet. Methods: Male C57BL/6 mice (8 weeks old) were divided into Control (C, n=18) fed with standard diet, and HFD (n=12) fed a 40% high fat diet for 15 weeks. Then, both groups were subdivided according to subcutaneous administration of LGT (200ug/kg/day) or vehicle for 5 weeks. Body weight, food and water intake were recorded weekly. Epididymal AT (EAT) was removed and weighed. Adipocyte density and volume were evaluated by haematoxylin-eosin staining, vascularization by fluorescence assay, PPARɣ expression by western blotting and GLP-1R, adiponectin and leptin mRNA levels were measured by RT-qPCR. Results: EAT mass was increased in HFD compared to C ($p<0.05$) and reduced in HFD+LGT ($p<0.001$ vs HFD). HFD also induced larger adipocyte volume ($p<0.01$ vs C) and lower adipocyte and vascular density ($p<0.01$ vs C), changes that were reversed in HFD+LGT ($p<0.01$ vs HFD). In HFD, PPARγ expression was decreased ($p<0.001$) while GLP-1R levels were elevated ($p<0.05$ vs C); HFD+LGT showed the opposite expression pattern ($p<0.05$ vs HFD). Adiponectin was reduced in HFD ($p<0.05$ vs C) and markedly increased in HFD+LGT ($p<0.001$ vs HFD). Leptin levels were decreased in both HFD and HFD+LGT compared to C ($p<0.001$). Conclusions: In obesity, LGT improves VAT structural changes and dysregulated adipokine expression, highlighting its potential as a therapeutic strategy to restore adipocyte functionality.



Monday, November 3, 2025

VIII International Congress in Translational Medicine

AREA | Microbiology and Immunology

POSTER TITLE | Development of a bioinformatic pipeline for identifying and characterizing operons associated with heavy metal resistance: Application to the epidemic clone Klebsiella pneumoniae ST258.

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POSTER ABSTRACT (350 WORDS MAXIMUM)

The use of bioinformatic tools is essential for analyzing the bacterial resistome. Although several programs are available to detect resistance genes, their identification becomes complex when these genes are part of operons, and most tools do not organize this information in a friendly way.
Currently, there is no automated and user-friendly bioinformatic tool for detecting heavy metal resistance (HMR) genes in Gram-negative bacilli. Their detection is important because they are found in the environment and act as a co-selection pressure for antimicrobial resistance. Furthermore, depending on the gene combination, different HMR profiles may be observed.
AMRFinderPlus is a software tool with its own database, available on GitHub, that identifies acquired resistance genes for antimicrobial, heavy metal, virulence factors, and biocide, as well as resistance-associated point mutations across several taxa.
Our objective was to develop a pipeline that integrates the local installation and execution of AMRFinderPlus, providing a friendly visualization of the resistome, the operon integration, and the prediction of expected phenotypes. Finally, we applied the tool to a case study.
We developed a Python3-based tool functioning in Jupyter-Notebook containing four main modules responsible for installing and running AMRFinderPlus locally. In this opportunity, the script was designed to analyze HMR operons for arsenic, silver, mercury, and telluric from the total prediction of AMRFinderPlus in multiple nucleotide assembled genomes. In the last step, the script generates a

summary table file along with a heatmap representing operon presence (purple)/absence(grey)/incomplete(yellow) by genome, able to be downloaded.
Thirty-two Klebsiella pneumoniae ST258 genomes were analyzed. The program properly detected the presence of the selected operons in 13/32 Kp-ST258 genomes. The detection was manually controlled using BLAST. The script allows users to modify the list of required genes according to their research needs.
The script was useful for multiple genome analyses, quickly integrating biological knowledge about operons and HMR, and presenting the results in a friendly manner. The pipeline versatility allows for adaptation to user needs, including new operons and/or particular genes of antimicrobial resistance or virulence.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Microbiology and Immunology

POSTER TITLE

Gut Microbiota and Telomere Length: Inflammatory Pathways Underlying Accelerated Cellular Aging in Obesity

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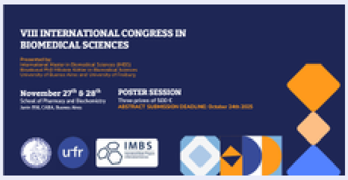
POSTER ABSTRACT (350 WORDS MAXIMUM)

The interaction between the gut microbiota (GM) and telomere length (TL) is a critical axis in the study of cellular aging, particularly in metabolic disease. This cross-sectional study aimed to analyze the GM composition and identify biomarkers linking the clinical-inflammatory profile to TL in individuals with obesity (OB) compared to controls. GM composition and diversity were evaluated by metagenomic

sequencing, and TL and inflammatory mediators (TLR4, IL-1β and NLRP3) were quantified by qPCR.

Principal Component Analysis captured the main separation between OB and Control groups along PC1, which accounted for 24.9% of total variance. This axis was defined as an Anthropometric-Metabolic Axis, driven by high loadings from BMI, circumferences, and metabolic markers (Insulin, HbA1c), while PC2 captured variance related to the lipid profile and GM diversity.
In the total sample, IL-1β expression is positively correlated with TLR4 receptor ($r=0.512$, $p<0.001$) and Waist Circumference (WC) ($r=0.262$, $p=0.036$), linking central adiposity to pro-inflammatory cytokine production. Activation of the inflammasome NLRP3, was correlated with Insulin levels ($r=0.386$, $p=0.038$), suggesting a role in insulin resistance. Regarding the GM, abundance of species F.prausnitzii, known for its butyrate production, correlated negatively with NLRP3 expression ($r=-0.338$, $p=0.022$), confirming its anti-inflammatory potential. Specifically in the OB group, TLR4 showed robust correlations with its effectors NLRP3 ($r=0.370$, $p=0.009$) and IL-1β ($r=0.543$, $p<0.001$). Furthermore, higher Shannon diversity correlated with lower IL-1β levels ($r=-0.397$, $p=0.022$) emphasizing the protective role of eubiosis. Crucially, NLRP3 expression correlated negatively with TL ($r=-0.459$, $p=0.037$), suggesting that chronic inflammasome activation drives telomere shortening and accelerated cellular aging in OB.

These findings establish that the activation of the TLR4/NLRP3/IL-1β inflammatory cascade is exacerbated in obesity, is linked to insulin resistance, and is inversely modulated by beneficial microbial markers (F.prausnitzii and Shannon diversity). The inverse correlation between NLRP3 expression and telomere length demonstrates that chronic inflammasome activation is a critical mechanism linking metabolic dysregulation and accelerated cellular aging in individuals with obesity.



Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Microbiology and Immunology

POSTER TITLE

Genomic insights into the resistance mechanisms of Achromobacter spp. in Argentina

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Achromobacter spp. are opportunistic pathogens increasingly associated with chronic respiratory infections, particularly in individuals with cystic fibrosis (CF) due to their persistence and intrinsic multidrug resistance. This study aimed to analyze species diversity, resistome, and mobilome in clinical isolates collected over nearly three decades in Argentina using a genomic approach.
A total of 82 Achromobacter spp. isolates obtained from patients treated in seven hospitals across

Argentina were analyzed. These isolates, collected between 1996 and 2024, were preserved in the collection of the Institute of Bacteriology and Molecular Virology (IBaViM) at the University of Buenos Aires. Whole-genome sequencing was performed using Illumina technology, and phenotypic antimicrobial susceptibility was assessed by minimum inhibitory concentration (MIC).
Ten species were identified, with A. xylosoxidans, A. ruhlandii, and A. insuavis being the most prevalent. A total of 56 sequence types (STs) were detected, including 30 newly identified ones. Species-specific OXA-type β-lactamases were found in A. xylosoxidans, A. ruhlandii, A. insuavis, A. dolens, and Achromobacter genogroup 20. Putative chromosomal class C β-lactamases were widely distributed, while AMZ was detected only in A. mucicolens and ELC exclusively in A. ruhlandii. AXC variants were identified in some A. xylosoxidans isolates and in most A. ruhlandii isolates. The efflux pumps AxyABM and AxyEF-OprN were present in all species, whereas AxyXY-OprZ, AxySUV, and AinCDJ were restricted to specific groups.
Nine different plasmid types were identified, including members of the IncP-1, IncP-6, rep5b, rep7a, and Col families. Insertion sequences from several families (Tn3, IS1, IS3, IS4, IS5, and ISNCY) were also detected, along with prophage regions, some of which were intact.
This study reveals a high degree of species and lineage diversity within the genus Achromobacter. The widespread detection of efflux pumps indicates a conserved, species-level resistome, while the presence of species-specific OXA enzymes, AxySUV, and AinCDJ suggests distinct resistance profiles among species. The identification of plasmids, insertion sequences, and prophages highlights the central role of mobile genetic elements in adaptation and persistence. The genomic characterization of this collection provides valuable insights into antimicrobial resistance mechanisms and the population structure of Achromobacter circulating in the region.

Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Oncology

POSTER TITLE

4-Methylumbelliferone Modulates the Peptidome and Reduces Apelin Expression in Chronic Myeloid Leukemia Cells

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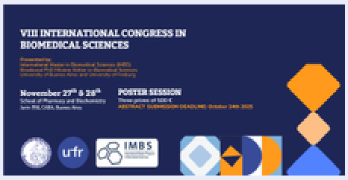
POSTER ABSTRACT (350 WORDS MAXIMUM)

Chronic myeloid leukemia (CML) is characterized by the presence of the Philadelphia chromosome, which

encodes a constitutively active tyrosine kinase known as BCR-ABL. Although treatment with tyrosine kinase inhibitors (TKIs) is highly effective, prolonged drug exposure exerts selective pressure that promotes the emergence of resistant clones, ultimately leading to therapeutic failure. This underscores the need to explore novel therapeutic strategies that enhance the efficacy of TKIs.
In this context, our group has previously demonstrated that 4-methylumbelliferone (4MU) sensitizes CML cells to imatinib. However, the molecular mechanisms underlying this chemosensitization remain unclear. The aim of this study was to further elucidate the mechanism of action of 4MU. To this end, we analyzed the peptidomic profile of CML cells treated with 4MU using MALDI-TOF mass spectrometry (MALDI-TOF-MS). The results revealed that 4MU differentially modulates several peptides between imatinib-sensitive and imatinib-resistant cells.
Among these peptides, one candidate was selected for validation by flow cytometry (FC), confirming that 4MU reduces apelin expression across the analyzed CML cell lines. Moreover, apelin treatment partially counteracted the effect of 4MU on metabolic activity, while an apelin antagonist sensitized the cells to imatinib (evaluated by XTT assay).
Taken together, our findings indicate that 4MU decreases apelin levels, suggesting that apelin downregulation may contribute to imatinib sensitization. These results highlight apelin as a potential therapeutic target and provide new insights into the translational mechanisms underlying 4MU activity in CML.



Monday, October 27, 2025

VIII International Congress in Translational Medicine

AREA

Oncology

POSTER TITLE

LORATADINE AND DESLORATADINE H1-ANTIHISTAMINES PROMOTE DNA DAMAGE, A CRUCIAL FACTOR IN THE RESPONSE TO THERAPIES, IN BREAST CANCER CELL LINES

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Breast cancer is the most frequently diagnosed cancer and the primary cause of cancer-related deaths worldwide in women. The need for innovative approaches to enhance the effectiveness of cancer therapy and prevent recurrence after initial treatment remains a priority. H1-antihistamines (HR1-A) are among the most ubiquitously used medications globally, affordable and safe. Retrospective epidemiological studies have reported an improved survival rate in women with breast cancer who used the HR1-A loratadine or desloratadine peri- or post-diagnosis, regardless of tumor stage or estrogen receptor levels. Preclinical research has demonstrated that two HR1-A compounds, which are not currently utilized in clinical settings, have shown antitumor effects. Our experiments on two breast cancer cell lines using loratadine and desloratadine, commonly prescribed in Argentina, resulted in reduced clonogenic survival (IC50c: 3–8 μM) and viability (IC50v: 30–65 μM). When these HR1-A were combined with 2Gy (a standard fraction dose in radiotherapy), there was a further enhancement in the radiotherapy-induced reduction in clonogenicity and viability.
The aim of this work was to investigate in MDA-MB-231 and MCF-7 breast cancer cell lines the effect of

loratadine and desloratadine (IC50c and IC50v) on DNA damage, a key factor in the response to ionizing radiation.
We performed the alkaline comet assay, a gel electrophoresis-based method that detects DNA single- and double-strand breaks and base damage in single cells. In both cell lines HR1-A higher concentration (IC50v) produced an increase in the DNA damage index after 3, 24 and 72 h of incubation. The lower concentration (IC50c) yielded the same results at 24 and 72 h. In addition, we determined by indirect immunofluorescence the formation of γ-H2AX foci, a marker of DNA double-strand breaks. The count of positive nuclei (harboring more than 4 γ-H2AX foci) showed that both HR1-A raised DNA damage at 24 and 72 h in both cell lines.
The results suggest that these commonly used antihistamines may sensitize tumor cells to different therapies by enhancing DNA damage, supporting further research on their potential as adjuvant agents in breast cancer therapy.

Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Oncology

POSTER TITLE

CYTOME ASSAY: A TOOL FOR A COMPREHENSIVE APPROACH TO CANCER

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Genomic instability plays a prominent role as an enabling characteristic for acquiring the hallmarks of cancer, yet relatively few studies have explored its contribution to cancer progression. In genetic toxicology, the presence of micronuclei (MN) in peripheral blood lymphocytes (PBL) is a cytogenetic biomarker of this phenomenon. It is currently validated as an early predictor of cancer risk and its clinical use is under study. The Cytome assay in PBLs, in addition to the frequency of MN in binucleated cells (BN), evaluates other biomarkers of genotoxicity (nucleoplasmic bridges -NPB-, nuclear buds -Buds-), cytotoxicity (%Viability) and cytostaticity (nuclear division index -IDN-). Our research team works on biomonitoring cervical cancer patients, and recently incorporated another population with a presumptive diagnosis of prostate cancer. Both types of cancer are different but share great relevance in public health, being among the five most diagnosed both worldwide and in Argentina. Following a protocol approved by the ethics committee of the Faculty of Pharmacy and Biochemistry of the UBA, informed consent and sociodemographic surveys are obtained, along with a sample of heparinised blood. To evaluate the impact of neoplastic severity, patients with cervical cancer are stratified according to the International Federation of Gynaecology and Obstetrics into low (stage I, n=46) and high (stages II-IV, n=54) grades. Higher-severity patients showed statistically significant ($p<0.01$, Mann-Whitney-U) differences in genotoxicity (MN/1000BN 11,3 [0,5-74,2] vs 27,9 [5,5-88,6] and NPB/1000BN 2,0 [0,0-27,0] vs 4,5 [0,0-43,8]), cytostaticity (IDN 1,24 [1,03-1,58] vs 1,14 [1,03-1,46]) and cytotoxicity (%Viab 97,3 [92,0-99,7] vs 93,4 [82,5-99,3]) biomarkers. Preliminary results from the first patients with a presumptive diagnosis of prostate cancer analysed (n=20) showed no statistically significant differences between those with and without neoplastic pathology. However, it is remarkable that the mean MN for both cases (48,0±13,1; 37,8±22,8) is above the reference range (0-30), consistent with an average age of 69±7 that implies high cumulative exposure (therapeutic, occupational, environmental). Our results highlight the usefulness of further clinical application of the Cytome assay in cancer patients. A greater understanding of the role of genomic instability in this disease will enable a better approach, reducing its incidence and mortality.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Oncology

POSTER TITLE

Melitherapy as a novel lipid-modulated strategy to regulate cell proliferation and motility.

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims:
Melitherapy is an emerging therapeutic strategy based on bioactive lipids to modulate the structure and functionality of the cell membrane. Since the plasma membrane is the first element in contact with the cell's environment, controlling its composition could allow us to modulate key cellular functions such as adhesion, communication, migration, and even cell proliferation or viability. The aim of this project is to evaluate new bioactive lipids as potential agents with antiproliferative and antimetastatic properties against different cancer cell types.

Methods:

This study evaluated the potential of melitherapy in two lung cancer cell lines: A549 and H1299. Two lipid-based compounds with specific structural and physicochemical properties were tested to compare their biological effects and identify features with greater therapeutic relevance. Cell viability assays were used to determine cytotoxic responses by flow cytometry using the Sytox Green reagent, while wound-healing and transwell assays were performed to evaluate cell migration and invasion following treatment. Wound closure was monitored and quantified using the Incucyte live-cell imaging system.

Results:
Our results demonstrated a dose-dependent decrease in cell viability within 24 hours of treatment. Regarding cell motility, transwell migration assays revealed a reduction in migratory capacity after 16 hours of treatment. Consistently, wound healing assays indicated a significant deterioration in migratory potential as a result of each treatment. Furthermore, the data revealed different effects depending on which bioactive lipid was used.

Discussion:
Our preliminary results suggest the effectiveness of melitherapy depends on the specific lipid composition, providing new insights into membrane-targeted strategies as innovative approaches to limit motility of lung cancer cells.

Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Pharmacology and Toxicology

POSTER TITLE

Co-delivery of ibuprofen and curcumin in nebulized polymeric micelles to optimize household air pollution adverse effects

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POSTER ABSTRACT (350 WORDS MAXIMUM)

It has been reported that the exposure to indoor particulate matter (PM) impairs redox metabolism and promotes inflammation, which might aggravate respiratory diseases. Lung epithelial cells are suggested to play a central role in this scenario, since they produce inflammatory and oxidative stress mediators following PM uptake. In the present work we aimed to study the pathways leading to redox metabolism alterations and NLRP3 inflammasome activation in A549 cells, EpiAlveolar 3D tissue and Balb/c mice exposed to indoor dust (ID) for up to 24 h. Additionally, this work explores a nano-pharmaceutical

approach to enhance the efficacy of anti-inflammatory and antioxidant drugs, focusing on curcumin (Cur) and ibuprofen (Ibu). These agents are encapsulated within Soluplus® micelles to form a novel drug delivery system. When A549 cells were exposed to ID, intracellular redox status and oxidative damage to lipids was observed after 24 h. Additionally, dose-and time-dependent NFkB nuclear translocation and NLRP3-inflammosome activation was evidenced after ID exposure. Consistently, we found an increase in IL-1β release. On EpiAlveolar 3D tissue model exposed to ID showed an impairment in barrier integrity together with an increase in HO-1 and 4-HNE signal. In addition, NLRP3 and ASC were also found in the 3D model as well as the increased IL-1β levels. The nanomicelles were meticulously characterized by drug loading, size distribution, morphological features and antioxidant capacity. Interestingly, preincubation with nanomicelles, containing Cur and Ibu on A549 cells, showed intracellular redox status and IL-1β levels similar to control group. The protective pathway seems to be involved with Nrf2 nuclear translocation.
On in vivo studies biodistribution with radiolabeled nanomicelles using 99mTc on Balb/c mice demonstrated the successful delivery of micelles mainly into the lungs after intranasal instillation. Moreover, nanomicelles revert the lung redox metabolism alterations due to the inhalation of ID particles on Balb-c mice, suggesting a protective effect potentially linked to its anti-inflammatory and antioxidant properties.
Our findings contribute to the understanding of the mechanisms by which ID promotes inflammation and oxidative stress in lung tissues and, overall, our nanoformulations stand as promising nanotechnological platforms to optimize lung therapy.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Pharmacology and Toxicology

POSTER TITLE

DESIGN AND CHARACTERIZATION OF BIOMOLECULE ENCAPSULATION SYSTEMS FOR TISSUE REGENERATION APPLICATIONS

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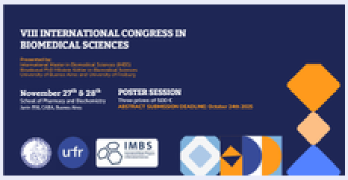
POSTER ABSTRACT (350 WORDS MAXIMUM)

Biopolymer hydrogels are effective carriers for bioactive molecules in wound healing and tissue regeneration. In this study, sodium alginate (SA) and carboxymethyl cellulose (CMC) were combined to form protein-encapsulating beads. Encapsulation efficiency (EE), influenced by bead crosslinking in $CaCl_2$ and molecular diffusion, was optimized by adjusting $CaCl_2$ viscosity using polyacrylamides of different molecular weights (40,000, 150,000, and 5,000,000 Da; types A, B, and C). Bovine serum albumin (BSA) was used as a model protein. Since polymer characteristics define EE, rheological characterization of SA.CMC, SA.CMC.BSA, and $CaCl_2$ solutions was performed to evaluate viscosity and viscoelastic behavior. Rheological analysis of SA.CMC and SA.CMC.BSA solutions showed shear-thinning behavior, SA.CMC.BSA being more viscous. Viscosity decreased with increasing temperature in both samples. Frequency sweep tests revealed viscous behavior (G" > G') at low frequencies and elastic behavior (G' > G") at higher frequencies. The crossover point (G' = G") occurred at 12.9 rad/s for SA.CMC and 6.31 rad/s for SA.CMC.BSA, indicating that the former has a weaker structural network and more liquid-like character. Viscosity analysis of $CaCl_2$ solutions with polyacrylamides A, B, and C showed similar viscosities for types A and B (2%), with B slightly higher. Type C exhibited much higher viscosity, so a lower concentration (0.5%) was used to match A and B.

BSA encapsulation was confirmed after dissolving the beads in Na_2CO_3 and analyzing the solution by SDS-PAGE to compare protein migration with a BSA molecular weight marker. Type B beads showed the highest EE (EE, 74.41 ± 4.19%) compared to types A and C (64.52 ± 1.34% and 58.34 ± 0.98%), likely due to the higher viscosity of the $CaCl_2$ solution. Swelling assays indicated that type B beads swelled the least over time, a desirable property for sustained drug delivery.
The combination of SA and CMC with polyacrylamide increased EE (32 ± 11.7% without polyacrylamide). Future work focuses on encapsulating anti-inflammatory cytokines, optimizing release kinetics and performing cytocompatibility assays.



Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA Pharmacology and Toxicology

POSTER TITLE IMT504 MODULATES MSC GENE EXPRESSION TOWARD REPAIR IN TYPE 1 DIABETES

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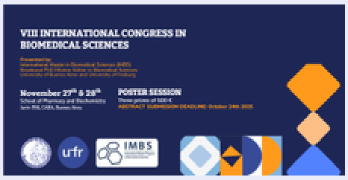
POSTER ABSTRACT (350 WORDS MAXIMUM)

Type 1 diabetes (T1D) features autoreactive T-cell–mediated β-cell loss and inadequate repair, calling for treatments that promote both immune control and regeneration. IMT504, a PyNTTTTGT immunomodulatory oligonucleotide, improves glycemia/insulitis in NOD mice and enhances mesenchymal stem-cell (MSC) function. Prior work also showed IMT504 favors regulatory T cells and downregulates pathogenic T-cell signaling genes (e.g.,PI3K/mTOR/STAT3/MAP3K7). Here, we aimed to define IMT504-driven MSC gene expression programs, with emphasis on cytokines linked to inflammation and repair.
Bone-marrow MSCs from pre-diabetic and diabetic NOD/Ltj mice were isolated and stimulated with IMT504 at the previously established effective dose for 2 h (short) or 18 h (prolonged), and gene expression was quantified by qPCR. Complementary assays (CFU-F proliferation, scratch-wound migration, and IMT504 subcellular localization) were performed for the biological context but are not the focus of this abstract.
IMT504 reprogrammed MSC transcription toward a reparative, anti-inflammatory profile. In MSCs from diabetic NOD mice, IMT504 decreased IL-6 and increased TGF-β expression compared to the control ($p < 0.05$), indicating a shift away from pro-inflammatory signaling and toward pathways that support tissue repair. In pre-diabetic MSCs, TGF-β remained stable while IL-6 changes were limited at the effective dose. These gene-level shifts are consistent with previously observed IMT504-associated gains in MSC proliferation and migration and with their cytoplasmic/nuclear localization pattern in MSCs.
IMT504 modulates MSC gene expression in the NOD model of T1D, favoring reduced IL-6 and elevated TGF-β—a profile compatible with anti-inflammatory, pro-repair activity. In the context of our previously presented T-cell findings (enhanced Treg programs and downregulation of pathogenic signaling genes), these results suggest that IMT504 integrates pro-regenerative MSC programming with immune-tolerance mechanisms. Together, the data supports IMT504 as a candidate to help preserve β-cell function and

mitigate autoimmune progression in diabetes.



Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA Pharmacology and Toxicology

POSTER TITLE NEONATAL EXPOSURE TO BISPHENOL A, BENZOPHENONES 2 AND 3 ALTER ESTROUS CYCLES AND GONADOTROPIN SUBUNIT GENE EXPRESSION LEVELS IN C57BL/6 MICE

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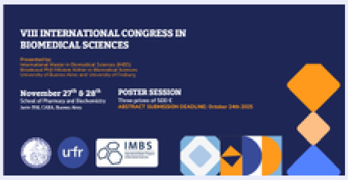
POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and aims:, (BPA), monomer of polycarbonate plastics, and Benzophenones (BPs), UV filters, are endocrine disrupting chemicals (EDC). Previously we described effects of the neonatal (Postnatal day (PND)1-PND10) exposure to BPA on the hypothalamic-pituitary-gonadal axis in female rats [1, 2]. Our aim was to evaluate the effects of the neonatal exposure to BPA, BP2 or BP3 on gonadotropin subunit gene expression levels in male and female C57BL/6 mice and estrous cycles in the females.
Methods: Pups were dosed orally, daily, from PND1-PND10, with BPA (50 µg/kg), BP2 or BP3 (250 µg/kg), or vehicle (control). Animals were weighted weekly. Age and weight at vaginal opening and estrous cycles were registered for the females. Males and females were sacrificed at PND50 (females were sacrificed in diestrus), pituitaries dissected and mRNA obtained using TriReagent (MRC Inc, USA). Gonadotropin subunit gene expression levels were measured by qPCR. Results were expressed as Media±SEM and analyzed by One-way ANOVA,
Results: Females exposed to all EDC had higher number of cycles over 14 days of cycling (ANOVA: $p<0.05$). Exposure to BPA had no effects on gonadotropin subunit gene expression in males (ns), whereas in females it increased Chorionic Gonadotropin Alpha-subunit (CgA), significantly decreased Follicle-stimulating hormone subunit beta (FSHbeta) ($p<0.05$) and slightly increased Luteinizing hormone subunit

beta (LHbeta), although this change was not significant (p=0.09). Exposure to BP2 significantly dereased CgA and FSHbeta gene expression levels in males (p<0.05), whereas it increased CgA in females (p<0.05), leaving the beta subunits unchanged. Exposure to BP3 increased CgA expression in males and females (p<0.05), and LHbeta subunit in the females (p<0.05).
Conclusions: Our results show that the exposure to EDCs present in plastics and personal care products alter parameters related to reproduction, in a sex-dependent manner. More studies are underway to further explore the effects observed.
Supported by: CONICET, ANPCyT, Int. Soc. for Neurochemistry, Fund. R. Barón, Fund. Williams, Fund. H. Bigand. (1.EHP 2009. 117(5): p. 757-762, 2.EHP 2010. 118(9): p. 1217-1222).



Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology and General Medicine

POSTER TITLE

IS ZINC SUPPLEMENTATION DURING GROWTH BENEFICIAL TO DIMINISH THE CARDIOVASCULAR AND METABOLIC ALTERATIONS OF METABOLIC SYNDROME?

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NO

Terms and Conditions

Accepted

POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and aims: Zinc supplementation may reduce metabolic and cardiovascular damage associated with metabolic syndrome, given zinc's antioxidant and anti-inflammatory properties. We aim to evaluate the effects of zinc supplementation on systolic blood pressure (SBP), intermediate metabolism and retroperitoneal adipose tissue (RPAT) in male Wistar rats fed a high-fat and fructose diet (HFFD) during post-weaning growth.
Methods: Male Wistar rats were fed from 21 to 81 days of life: control diet (CC, Zinc 30 ppm), zinc supplemented diet (CZ: Zinc 190 ppm), high-fat diet (CFF; 60% pork fat calories, 30 ppm Zinc) and 10% fructose in drinking water, high-fat diet supplemented with zinc (ZFF; 60% fat calories, 190 ppm Zinc) and 10% fructose in drinking water. SBP, oral glucose tolerance test (OGTT), lipid profile, and adipokine ARNm expression, morphological changes and oxidative state in RPAT were evaluated at 81 days. Statistics: 2-way ANOVA, post-hoc test: Bonferroni: *p<0.05 vs CC; +p<0.05 vs CFF; &p<0.05 vs CZ; n=9 per group.
Results: ZFF rats showed reduced SBP, insulin resistance, triglyceridemia, and cholesterol compared to CFF. ZFF rats showed reduced adipocyte hypertrophy (CC:5258±312; CZ: 3070±208*; CFF:7203±454*; ZFF: 5368±549+,& µm2) and media layer area/light area of vessels (CC:2,2±0,4; CZ:3,1±0,5; CFF:4,8±0,4**; ZFF:2,7±0,3++), lower thiobarbituric acid reactive species concentration (CC:0,21±0,01; CZ:0,09±0,02*; CFF:0,38±0,03*; ZFF:0,23±0,04+,& nmol/mg protein), higher antioxidant superoxide dismutase activity (CC:5,8±0,3; CZ:12±2*; CFF:6,4±0,7; ZFF:11±2+ uSOD/mg protein) and NRF2 mRNA expression (CC:2,3±0,4; CZ:8,9±1.1*; CFF:1,3±0,2; ZFF:4,6±0,7+,&).
Conclusion: Zinc supplementation during growth reduces cardiovascular and metabolic damage induced by HFFD. In CZ rats, zinc improves insulin resistance, glycemia, cholesterol levels, oxidative stress in RPAT.

Thursday, October 23, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

METABOLIC ENVIRONMENT MODULATES THE CARDIOPROTECTIVE EFFICACY OF STEVIOSIDE AGAINST ISCHEMIA-REPERFUSION INJURY

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THE POSTER APPLIES FOR PRIZE

NO

Terms and Conditions

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and aims: The cardiac metabolic environment is a critical determinant of the mitochondrial function and response to ischemia-reperfusion (Is-Rs) injury. Fasting redirects metabolism toward fatty acid oxidation, providing a relevant model to test this influence. Stevioside (S), a natural glycoside, has shown cardioprotective potential against Is-Rs, but its efficacy across metabolic states is unclear.

Therefore, this study aimed to evaluate how fasting impacts the cardioprotective, mitochondrial, and ultrastructural actions of S in rat hearts subjected to Is-Rs.
Methods: Female Sprague-Dawley rats received S (168 mg/kg/day, 15 days) or vehicle (Control, C). Animals were randomized into fed (C, S) and 24h-fasted (CF, SF) groups (n=5/group). Isolated hearts (Langendorff) underwent 25 min global ischemia and 60 min reperfusion. We measured rate-pressure product (RPP), contraction and relaxation velocities (±dP/dT), left-ventricular end-diastolic pressure (LVEDP), and infarct size. In mitochondria energized with pyruvate/malate or succinate, we assessed ATP synthesis, respiratory complex activities, calcium retention capacity (CRC), and membrane potential (Δψ). Baseline echocardiography and transmission electron microscopy (TEM) were performed. Statistics used ANOVA followed by Tukey's test ($p<0.05$).
Results: Echocardiography showed no baseline differences. At 5 min of reperfusion, stevioside improved RPP recovery in S and SF ($p<0.05$ vs C/CF), but in SF the benefit was transient and disappeared by 10 min. Fasting eliminated the differences in ±dP/dT and LVEDP observed between S and C. Stevioside reduced infarct size in S ($p<0.05$) but not in SF. Fasting itself altered pre-ischemic mitochondrial function, markedly decreasing ATP synthesis, tissue ATP levels and respiratory complex activities ($p<0.001$ vs C and S). Interestingly, fasting increased pre-ischemic CRC ($p<0.001$) and preserved it better after Is-Rs in SF, while eliminating pre-ischemic Δψ differences between S and C. TEM showed S hearts with better-preserved sarcomeres and mitochondria organization versus controls, whereas SF hearts exhibited significant ultrastructural changes, including mitochondrial clustering and vacuolization.
Conclusions: Stevioside's cardioprotective efficacy is strongly dependent on metabolic background. Fasting-induced changes altered mitochondrial function and ultrastructure, weakening the beneficial effects of stevioside seen in the fed state. These findings underline the importance of metabolic context when evaluating cardioprotective strategies.

Friday, October 31, 2025

VIII International Congress in Translational Medicine

AREA	Physiopathology and General Medicine
POSTER TITLE	TIMING IS EVERYTHING: HOW HYPOTHYROIDISM IMPACTS CARDIOVASCULAR HEALTH IN ADULTHOOD
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Terms and Conditions	Accepted

POSTER ABSTRACT (350 WORDS MAXIMUM)

The aim of this study was to investigate whether hypothyroidism induced by methimazole (MMZ) at different stages of life (fetal, peri and postnatal) affects cardiovascular function in adulthood. Pregnant Sprague-Dawley rats were divided into three groups according to the time of MMZ exposure: G (MMZ 0,02% in drinking water from day 9,5 of gestation until parturition), GL (MMZ 0,02% from day 9,5 until 21 days after parturition), and C (control, tap water). After weaning, all male cubs received a balanced diet

and water ad libitum, and were assigned to group A (from G), group B (from GL), group C (control), and group D (from C, treated postnatally with MMZ 0,02%). Thyroid state was assessed to confirm treatment efficacy. At 90 days of age, systolic arterial pressure (PAS) and echocardiographic parameters were measured, including heart rate (HR), left-ventricular internal diameter, anterior and posterior wall thickness, ejection fraction, and fractional shortening. Isolated cardiomyocytes were analyzed for cell shortening, calcium transients, relaxation, and calcium reuptake at 3 Hz. Antioxidant enzyme activities of superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx) were quantified. Data is expressed as Mean ± SEM and analysed using one-way ANOVA (SPSS v22.0, $p < 0,05$ vs. C). Compared with controls, plasma T3 and T4 levels increased in groups A and B respectively, but decreased in D. TSH was reduced only in D. PAS was unchanged, while HR decreased in D. All cardiovascular parameters were decreased in D, with no alterations in A or B. Cardiomyocyte shortening and relaxation were altered in B and D, and calcium transients decreased exclusively in D. All groups showed increased calcium reuptake times. SOD activity increased in all groups, catalase increased in A but decreased in D, and GPx remained unaltered. These results demonstrate that the timing of thyroid disruption is critical for determining long-term cardiovascular outcomes. Congenital and early-life hypothyroidism appears to elicit compensatory mechanisms mediated by maternal hormones, whereas postnatal hypothyroidism leads to structural and functional cardiac impairment. The up-regulation of antioxidant enzymes, particularly SOD, may represent an adaptive cardioprotective response.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

Modulating dual effects of TNF-α to enhance dopaminergic progenitor viability

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Terms and Conditions

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Parkinson's Disease (PD) is characterised by the progressive loss of dopaminergic neurons (DA) of the substantia nigra pars compacta. There are five on-going clinical trials using transplanted pluripotent stem cell-derived dopaminergic progenitors (PS-DA) for PD. However, low cell survival (0,5-10%) is a main limitation and hinders standardization. We and other have found that inflammation can alter DA viability. Therefore, we studied the inflammatory response elicited by cell transplantation and how this affected the survival of transplanted PS-DA.

We have obtained and characterized PS-DA and found early and transient neutrophil recruitment between 1 to 7 days after striatal PS-DA transplantation in immunosuppressed rats. We observed astro- and microgliosis from day 1 to 28 after transplantation of human PS-DA into the striatum. The latter was associated with the expression of tumor necrosis factor alpha (TNF). In vitro, microglial conditioned media secreted TNF and decreased the number and neurite length and increased apoptosis of PS-DA at immature and mature states. Etanercept, a systemic TNF inhibitor, reverted the first two effects. Moreover, there was a decrease in PS-DA viability when incubated with TNF and Actinomicin D at a mature stage. On the other hand, we have shown that TNF can have dual effects on DA viability depending on which receptor is used (TNFR1 or TNFR2), since only TNFR1 produces neurodegeneration. We found that dopaminergic progenitors expressed TNFR1 by immunofluorescence and RNASeq.
Our results suggest that transplanted human PS-DA activates microglia which secretes TNF. In turn, TNF is toxic to PS-DA. Our data indicate that a selective inhibition of TNFR1 could increase cell survival after PS-DA transplantation.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA	Physiopathology
POSTER TITLE	Redox-dependent aggregation of IgG induced by Cu(II) and reducing agents
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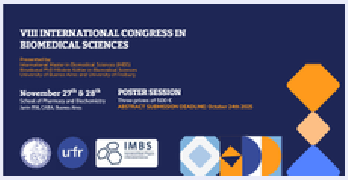
POSTER ABSTRACT (350 WORDS MAXIMUM)

Introduction: Cu(II) induces the aggregation of IgG, an effect strongly potentiated by ascorbic acid (AA) but not observed in other plasma proteins such as albumin. Given that both Cu(II) ions and high AA concentrations coexist in plasma, this phenomenon may modulate the levels of circulating protein aggregates. Such protein aggregates have been reported in physiological and pathological conditions and are known to increase with age. Objectives: To understand the mechanism of Cu(I)-induced irreversible

aggregation of IgG. Results: Cu(II)-induced IgG aggregation, evidenced by increased opalescence (405 nm), is potentiated by AA and other reducing agents such as N-acetylcysteine, cysteine, and dithionite ($p<0.001$), whereas reducing agents alone do not promote aggregation. The aggregated fraction comprises monomeric, dimeric, and multimeric IgG species as observed by SDS-PAGE. The O_2 consumption rate of the Cu(II)/AA system at pH 7.4 decreases significantly in the presence of IgG ($p<0.001$). After metal chelation with EDTA or GSH, about 60% of the aggregate remains ($p<0.001$). The employement of dithionite which reduces Cu(II) to Cu(I) but also depletes the O_2 in the system causes a nearly complete loss of turbidity after the addition of the chelating agents ($p<0.001$). Concomitant with O_2 consumption, free thiols (–SH) are generated and detected spectrophotometrically with Ellman's reagent ($p<0.01$). This reagent covalently modifies the reduced –SH groups, thus preventing their involvement in new disulfide bonds. However, their covalent modification does not prevent disaggregation after metal chelation. Furthermore, the –SH groups remain detectable throughout the aggregation process. Discussion: These findings indicate that reducing agents potentiate IgG aggregation in the presence of Cu(II) through Cu(I) formation independently of O_2-derived species, since dithionite also induces aggregation. However, O_2 determines the irreversibility of the process: aggregates formed under aerobic conditions resist EDTA dissolution, while those formed under O_2 depletion are fully reversible. Therefore, Cu(II)/AA redox cycling may cleave one or more disulfide bonds in IgG, generating –SH groups, but these are not responsible for irreversible aggregation. Overall, Cu(II) and reducing agents promote IgG aggregation through a redox-dependent mechanism in which O_2 dictates the reversibility of the aggregates independently of disulfide bond cleavage.



Thursday, October 30, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology and General Medicine

POSTER TITLE

Assessing Residual Cardiovascular Risk through Lipid and Inflammatory Profiles in Coronary, Obese, and Control Subjects

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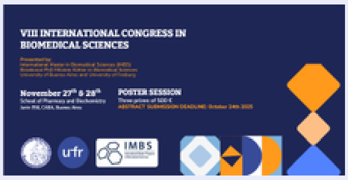
POSTER ABSTRACT (350 WORDS MAXIMUM)

Introduction:
Patients with established cardiovascular disease (CVD) often display residual risk (RR) even after achieving target low-density lipoprotein cholesterol (LDL-C) levels. Beyond LDL-C, remnant cholesterol (RLP-C) and non-HDL cholesterol (nonHDL-C) have emerged as key contributors to this risk. Obesity, a chronic pro-inflammatory condition, exacerbates atherogenic dyslipidemia and amplifies cardiovascular risk. Identifying metabolic and inflammatory markers associated with RR in obese individuals without CVD and in patients with established CVD could improve both primary and secondary prevention strategies.
Objective: To compare the metabolic and hematimetric inflammatory markers among healthy controls, obese subjects, and patients with stable CVD, in order to evaluate their contribution to residual cardiovascular risk.
Methods: We analyzed 41 healthy normal-weight controls, 34 obese subjects without clinical CVD, and 62 patients with stable CVD (30% obese, 37% overweight), all receiving lipid-lowering therapy. Total cholesterol (TC), triglycerides (TG), LDL-C, HDL-C, apolipoprotein B (ApoB), fasting glucose, and hematological parameters were determined by automated methods. Derived indices included remnant cholesterol (RLP-C), nonHDL-C, TG/HDL-C ratio, neutrophil-to-lymphocyte ratio (NLR), and systemic inflammation index (SII = NLR × platelets). The study was approved by the institutional bioethics committee (Resolution No. 1306-25).
Results: TG and RLP-C levels were higher in obese subjects (203 [151–298] and 29 [13–56] mg/dL, respectively) than in CVD patients (115 [37–338] and 19 [3–82] mg/dL) and controls (68 [37–158] and 12 [1–30] mg/dL) ($p < 0.001$). Patients with CVD presented lower HDL-C, LDL-C, and ApoB levels ($p < 0.001$) but higher RLP-C, glucose, and TG/HDL-C ratios ($p < 0.001$) compared with controls. Both CVD and obese subjects exhibited higher NLR (2.5 [1–8.8] and 2.4 [0.7–5.4], respectively), RDW (13 [10.9–25.9] and 12.8 [11.9–14.8]) compared to controls (1.7[0.9–3.7] and 12.5[11.6–14.3])($p < 0.05$). The SII was highest in obese subjects (703 [301–1372]) compared to CVD patients (528 [182–1731]) and controls (476 [216–1247]) ($p < 0.05$).
Conclusions: While lipid-lowering therapy normalizes traditional lipid panel in CVD patients, elevated RLP-C and persistent inflammation mirror findings in obesity, underscoring the existence of residual cardiovascular risk not reflected by standard lipid parameters alone.



Monday, October 27, 2025

VIII International Congress in Translational Medicine

AREA

Physiopathology

POSTER TITLE

KUPFFER CELLS AS THERAPEUTIC TARGETS IN STEATOHEPATITIS: EFFECTS OF MACROPHAGE DEPLETION VERSUS HO-1-MEDIATED FUNCTIONAL REPROGRAMMING

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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and Aims: Inflammation driven by Kupffer cells (KCs) is critical for the progression of metabolic dysfunction-associated steatohepatitis (MASH). Previous results from our laboratory indicate that administering 30% sucrose in the drinking water (SRD) to male rats for 12 weeks induces the development of steatohepatitis without fibrosis, accompanied by both peripheral and hepatic IR and dyslipidemia. The aim of this study was to investigate the role of KCs in SRD-induced MASH by comparing the effects of direct KC depletion versus functional reprogramming by the induction of Heme Oxygenase-1 (HO-1).
Methods: Male Wistar rats were fed SRD (30% sucrose water) and a standard solid diet “ad libitum” for 12 weeks. On the 10th week, SRD subgroups received $GdCl_3$ (10 mg/kg/72h i.p.) or hemin (15 mg/kg/48h i.p.) up to the 12th week. We analyzed hepatic histology (NAS score) and inflammatory gene expression. Isolated non-parenchymal fractions and determined HO-1/KC co-localization by immunofluorescence.

Systemic/hepatic insulin sensitivity, oxidative stress, and apoptosis markers were also assessed.
Results: GdCl3 treatment effectively reduced KC markers and pro-inflammatory gene expression. This effect correlated with attenuated hepatocyte ballooning, oxidative stress, and apoptosis. GdCl3 also improved peripheral insulin sensitivity, with no effects on local metabolism. Although hemin treatment did not affect KCs levels, it robustly induced HO-1 specifically within this population. This functional modulation also reduced M1 markers, ballooning, oxidative stress, and cell death. However, unlike GdCl3, hemin treatment also restored hepatic insulin sensitivity, activated fatty acid oxidation, and restored proper mitochondrial dynamics and autophagy.
Conclusions: KCs are key drivers of inflammation and injury in SRD-induced MASH development. Both KC depletion and HO-1-mediated functional reprogramming ameliorated KC-mediated injury. However, inducing HO-1 in KCs confers additional, superior benefits by also restoring hepatic metabolic function. This suggests KCs play a role beyond just responding to pathogens or injury and restoring their physiological function via HO-1 induction hints at a potential physiological contribution to metabolic balance that is lost or corrupted during MASH.

Friday, October 24, 2025

VIII International Congress in Translational Medicine

AREA	Physiopathology
POSTER TITLE	Cardioprotective mechanisms against ischemia/reperfusion injury in experimental dyslipidemia
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POSTER ABSTRACT (350 WORDS MAXIMUM)

Background and aims: Ischemic postconditioning (IP) reduces infarct size and mitigates ischemia/reperfusion (I/R) injury. However, we previously demonstrated that a high-fat diet (HFD) abolishes this cardioprotective effect, likely due to redox imbalance. This study aimed to evaluate whether N-acetylcysteine (NAC) can restore the cardioprotective effect of IP by reducing infarct size and reestablishing redox balance during the early stages of atherosclerosis. Methods: Male C57BL/6 mice were assigned to two dietary groups: control diet (CD) or high-fat diet (HFD). During the last three weeks of feeding, a subgroup of animals received NAC (10 mM) in the drinking water. After completing the protocol, hearts were subjected to the ischemia-reperfusion procedure. Lipid profiles, hepatic histology, and GSSG/GSH ratios were also assessed. Results: NAC treatment significantly reduced infarct size in both CD- and HFD-fed mice (HFD-I/R-NAC: 39.7 ± 4.5% vs. HFD-IP-NAC: 26.4 ± 2.0%, $p < 0.05$, $n = 7$), concomitant with increased GSH activity. NAC also improved the lipid profile, with a marked rise in HDL-cholesterol levels (CD: 68.4 ± 3 mg/dL; HFD: 79.9 ± 3 mg/dL; CD-NAC: 79.2 ± 5 mg/dL; HFD-NAC: 103 ± 5 mg/dL; $n = 7$) and counteracts the increase in vacuoles induced by an HFD. Conclusions: N-acetylcysteine restores the cardioprotective effect of ischemic postconditioning in mice fed a high-fat diet, reducing

infarct size and improving redox homeostasis, lipid profile and liver histology. These findings suggest that redox balance plays a critical role in preserving IP-induced cardioprotection and highlight NAC as a potential therapeutic strategy for mitigating diet-related loss of myocardial resistance to I/R injury.