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CONFERENCE ABSTRACTS

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New generation of pharmaceutical scientists

Clinical Pharmacy training through an academic league: an experience report from a Brazilian public university

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Background: Clinical Pharmacy education has become a strategic pillar in strengthening patient-centred care. In this context, academic leagues have emerged as complementary educational spaces, capable of integrating teaching, research and community engagement while fostering the development of clinical competencies during undergraduate training. The Academic League of Clinical Pharmacy and Pharmaceutical Care at the Federal University of Paraíba (LAFARCLIN-UFPB), founded in 2020 by Pharmacy students and faculty members, represents a structured initiative within the Health Sciences Centre. This study describes the experience of LAFARCLIN-UFPB in the training of Pharmacy students.

Method: This study presents an experience report of the activities developed by LAFARCLIN-UFPB, a non-profit civil organisation of unlimited duration, based at the Department of Pharmaceutical Sciences, Federal University of Paraíba (UFPB), Campus I, João Pessoa, Brazil. The league is composed of undergraduate students and academic supervisors, with its activities organised into three core pillars: teaching, research and community engagement.

Activities include clinical case discussions, thematic seminars, training in clinical methods, scientific production and health screening initiatives targeting chronic non-communicable diseases, such as hypertension, diabetes, dyslipidaemia and asthma.

Within these initiatives, students and professionals deliver clinical services, including health education, and perform procedures such as blood pressure measurement, capillary blood glucose testing, waist circumference assessment and bioimpedance analysis.

All activities are planned annually and conducted through regular meetings under academic supervision.

Results: LAFARCLIN-UFPB has contributed to the development of clinical competencies, critical thinking and evidence-based decision-making among participating students. The activities facilitated the integration of theory and practice, encouraged student engagement and promoted interprofessional experiences.

Additionally, the league strengthened scientific production, with participation in conferences and the development of academic outputs. In the domain of community engagement, health education initiatives enhanced links with the community and reinforced the university's social impact.

Conclusion: The experience of LAFARCLIN-UFPB highlights the potential of academic leagues as a complementary strategy in Clinical Pharmacy education. By promoting the integration of teaching, research and community engagement, such initiatives contribute to the development of essential professional competencies. The replication of similar models may strengthen pharmacy education and expand the role of pharmacists in clinical settings.

Role of estrogen signaling in the development of chemotherapy-induced peripheral neuropathy

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Background: Antitumor therapy alters endogenous hormones . Estrogen is an important hormone that affects the body and supports mental and physical health. Recently, estrogen was shown to play a role in regulating pain. However, the relationship between estrogen levels and the incidence of chemotherapy-induced peripheral neuropathy (CIPN) remains unclear.

Purpose: We analyzed the JMDC medical database to show the effects of estrogen- related medicines on CIPN. Furthermore, in a preclinical study, we assessed the effects of reducing estrogen levels on CIPN.

Method: Patients administered platinum-, taxane-, or vinca-alkaloid-based anticancer medicines were selected from the JMDC claim database. We calculated the incidence of neuropathy using estrogen agonists/antagonists or tumor species. In a preclinical study, C57BL6 female mice (7 weeks old) underwent bilateral ovariectomies under anesthesia. One week after the operation, mechanical and thermal sensitivities were analyzed using the von Frey, acetone, or capsaicin tests. Oxaliplatin (6 mg/kg/day) was administered intraperitoneally once a week.

Results: Antiestrogen medication increased the incidence of CIPN in patients with gastrointestinal and lung cancers. Moreover , CIPN was exacerbated by estrogen agonists in patients with gastrointestinal, liver, and lung cancers. Ovariectomized mice showed significantly exacerbated nociceptive behavior compared with sham mice without anti-tumor medicine treatment. Nociceptive behavior was exacerbated in ovariectomized mice after oxaliplatin treatment.

Conclusion: Our results suggest that estrogen regulates homeostasis in peripheral neurons and that fluctuations in estrogen levels change the nociceptive threshold. Estrogen-related medicines used in clinical practice may require careful monitoring during anticancer therapy.

Advances in carbon nanomaterial-based sensing and amplification: New horizons in diagnostics of gene-related diseases

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Background: Recent breakthroughs in nanotechnology have launched a new epoch of nucleic acid diagnostics and disease surveillance. Carbon nanomaterials (CNMs), a promising class of agents in gene analysis, have emerged as powerful enhancers in both nucleic acid sensing and amplification. This study aims to explore the advantages of CNMs in clinical applications and propose actionable solutions to address the key challenges ahead.

Method: More than fifty studies involving CNMs in nucleic acid sensing and amplification were reviewed. The characteristics of each method were comprehensively analyzed to identify favorable properties and limitations of CNMs.

Results: The surface of CNMs can be functionalized with various biological molecules and integrated with diverse nucleic acid and signal amplification techniques to enhance sensitivity and selectivity in the quantification of DNA and RNA. Thanks to their unique properties—such as biocompatibility, functionalization versatility, and low toxicity—CNMs have enabled extremely low detection limits in both optical and electrochemical sensing strategies. Moreover, CNMs can directly enhance nucleic acid amplification when specific nanomaterials are incorporated. By minimizing non-specific annealing and suppressing mismatched primer formation, CNMs significantly improve the efficiency and specificity of PCR and other amplification methods, thereby expanding their utility in gene analysis and precision medicine. However, critical challenges such as batch-to-batch variability and limited scalability highlight areas for future investigation. Emerging trends, including miniaturization, multiplexing, and the integration of smart technologies, offer exciting new directions for CNM-based diagnostics.

Conclusion: Supported by growing evidence, CNMs demonstrate remarkable potential in nucleic acid sensing and amplification. Nevertheless, key obstacles must still be overcome to facilitate the transition from laboratory research to clinical application. With continued collaboration between scientists and healthcare professionals, CNMs are poised to play a pivotal role in advancing personalized medicine and

improving access to precision diagnostics in the near future for patients in need.

Targeting the mitochondrial SLC25A39–ALDH7A1 redox circuit to restore cardiac resilience: A novel circRNA-LNP therapeutic strategy for diabetic cardiomyopathy

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Background: Diabetic cardiomyopathy (DCM) remains a leading cause of heart failure in metabolic syndrome, driven by mitochondrial dysfunction, oxidative stress, and energy depletion. Current therapies primarily manage systemic glucose homeostasis but fail to address the underlying mitochondrial redox imbalances that perpetuate cardiac injury. The mitochondrial inner-membrane transporter SLC25A39 has recently emerged as a critical regulator of glutathione (GSH) import; however, its role in cardiac pathophysiology remains unexplored. This study identifies a novel SLC25A39-dependent mitochondrial redox circuit whose disruption triggers ferroptosis and cardiac failure, and demonstrates the therapeutic potential of its restoration using a circRNA-LNP delivery system.

Method: DCM was established in male C57BL/6J mice using a high-fat diet and streptozotocin. Cardiomyocyte-specific SLC25A39 knockdown (KD) and overexpression (OE) were achieved via AAV9 vectors under the cTnT promoter. Cardiac function was assessed by echocardiography, mitochondrial morphology by transmission electron microscopy, and ferroptosis by lipid peroxidation (MDA), iron deposition (Prussian Blue), and GPX4/FTH1 expression. Proteomic analysis and co-immunoprecipitation identified SLC25A39-interacting proteins, with ALDH7A1 functionally validated via cycloheximide-chase assays and dual-gene manipulation. Therapeutic efficacy was evaluated using a circRNA-LNP system co-expressing SLC25A39 and Tim22 (circSLC25A39-Tim22).

Results: This study identifies SLC25A39 as a critical regulator of mitochondrial redox homeostasis in diabetic cardiomyopathy (DCM). SLC25A39 expression was markedly downregulated in diabetic myocardium and in cardiomyocytes exposed to metabolic stress, correlating with mitochondrial fragmentation and disorganization. Cardiomyocyte-specific SLC25A39 knockdown provoked profound mitochondrial structural damage, including cristae dissolution and membrane rupture, accompanied by global suppression of oxidative phosphorylation complexes and significant contractile dysfunction. These changes were coupled with hallmark features of ferroptosis—elevated lipid peroxidation, iron accumulation, and depletion of mitochondrial GPX4 and FTH1—demonstrating that SLC25A39 loss licenses ferroptotic cell death. Conversely, AAV9-mediated SLC25A39 restoration preserved mitochondrial architecture, recovered OXPHOS complex expression, normalized membrane potential, and improved cardiac function while suppressing ferroptotic markers. Mechanistically, proteomic analysis identified ALDH7A1 as a direct SLC25A39-interacting partner. SLC25A39 stabilized ALDH7A1 protein, prolonging its half-life and sustaining mitochondrial NAD^+/NADH balance. This functional coupling was essential, as dual knockdown of both proteins abrogated the protective effects, whereas ALDH7A1 overexpression partially rescued SLC25A39 deficiency. Notably, a circRNA-LNP system co-expressing SLC25A39 and its mitochondrial integrator Tim22 achieved sustained dual-gene delivery, demonstrating superior therapeutic efficacy compared to SLC25A39 alone. This combinatorial strategy fully restored OXPHOS complexes, normalized NAD^+ redox balance, and maximally suppressed ferroptosis and fibrosis.

Conclusion: This study establishes SLC25A39 as a master regulator of a mitochondrial redox circuit coupling glutathione import to NAD^+ homeostasis via ALDH7A1 stabilization, thereby protecting the diabetic heart from ferroptotic injury. By demonstrating that targeted restoration of the SLC25A39–ALDH7A1 axis reverses mitochondrial dysfunction and cardiac failure, we identify this pathway as a compelling therapeutic gateway. The circRNA-LNP-mediated co-delivery of SLC25A39 and Tim22 represents a promising precision medicine strategy for DCM, reframing the disease as a targetable disorder of mitochondrial circuit failure.

Mapping the Portuguese pharmaceutical workforce abroad: Findings from the First National Diaspora Census

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Background: The international mobility of pharmacists has grown significantly, and it is estimated that more than 1,200 Portuguese pharmacists are currently working abroad, out of a total of 17,306 registered with the Portuguese Pharmaceutical Society. However, structured and

comprehensive data on this population remain scarce. To address this gap, the Portuguese Pharmaceutical Society established the Pharmaceutical Diaspora Council and launched the first Census of the Portuguese Pharmaceutical Diaspora to characterise pharmacists' demographic profiles, geographic distribution, professional pathways, motivations, and expectations.

Purpose: This initiative aims to generate a structured overview of Portuguese pharmacists working internationally and to support the development of strategic initiatives that strengthen connections between the diaspora and the national professional community. By leveraging international experience and advanced competencies acquired abroad, the census seeks to inform professional development strategies, promote the integration of global best practices, and ultimately enhance the quality and impact of pharmaceutical services in Portugal.

Method: The census was conducted between 10 October and 16 November 2025 using a voluntary online questionnaire distributed through the communication channels of the Portuguese Pharmaceutical Society. The questionnaire included sections on identification, academic education, professional history, current country of practice, motivations for migration, relationship with the Portuguese Pharmaceutical Society, and expectations regarding support and engagement.

Quantitative data were analysed using descriptive statistics, including frequency distributions and mean scores for Likert-scale responses. Qualitative data from open-ended questions were analysed thematically to identify recurring themes. All responses were anonymised and processed in accordance with institutional confidentiality and data protection policies. As analysis is ongoing, the results presented reflect data assessed at the time of submission.

Results: Among the 134 respondents, the findings reveal a geographically diverse diaspora distributed across Europe, the Americas, Asia and Oceania, with the largest clusters being located in the United Kingdom, Switzerland, Norway, France, Germany, the Netherlands and Ireland. Professional roles span community and hospital pharmacy, the pharmaceutical industry, academia and research, with a strong concentration in areas such as regulatory affairs, quality, pharmacovigilance, market access and clinical pharmacy.

Key drivers for migration include career progression opportunities, higher remuneration, greater professional recognition and access to advanced training or research environments.

Regarding intentions to return to Portugal, most respondents do not exclude this possibility but emphasise that it depends on improvements in career progression pathways, professional recognition and working conditions. Qualitative feedback highlights persistent gaps in clinical pharmacy development, recognition of foreign qualifications and career mobility, influencing both emigration decisions and considerations about return. Respondents also expresses strong interest in maintaining engagement with the Portuguese Pharmaceutical Society, particularly through support for international qualification recognition, mobility guidance, continuing education, mentorship and professional networking.

Conclusion: This first census provides a comprehensive overview of the Portuguese pharmaceutical diaspora. The findings highlight a highly qualified, globally dispersed workforce whose professional trajectories reflect both international opportunities and structural challenges within Portugal. These insights will support the development of strategic initiatives by the Portuguese Pharmaceutical Society to strengthen engagement, facilitate professional mobility, and leverage the diaspora's expertise for the advancement of the profession.

A new pharmaceutical paradigm: integrating the administration of vaccines and injectable medicines as an intrinsic competence of pharmacists

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Background: The Portuguese Pharmaceutical Society (PPS) recognises and certifies pharmacists' competences in line with their professional responsibility and contribution to public health. In Portugal, the administration of vaccines and injectable medicines (AVIM) in community pharmacies is available nationwide, with more than 7,700 pharmacists qualified to provide this service (approximately 44% of all pharmacists). The sustained growth of the number of competent professionals highlights the contribution of pharmacists to public health and supports the recognition of AVIM as an intrinsic competence of the pharmacy profession. Moreover, pharmacists with this competence contribute significantly to the Portuguese healthcare system by easing its workload and expanding access to services such as vaccination in the community.

Given the consistent and robust implementation of this competence in professional practice, together with the significant increase in the number of qualified pharmacists, it is now both timely and appropriate for AVIM to evolve into a core, entry-level competence for all pharmacists.

Until 2026, PPS recognition of competence in AVIM depended on completion of an PPS-recognised course in vaccine and

injectable medicine administration, together with training in basic life support, undertaken after registration with the PPS.

In response to a PPS initiative, universities formally integrated AVIM training into the curriculum of the Integrated Master's Degree in Pharmaceutical Sciences (MDPC). From the 2025/2026 academic year onwards, students began receiving theoretical and practical training in AVIM prior to their curricular placement.

Also, the role of the clinical placement has been strengthened, now incorporating the AVIM training into supervised practice. From 15 March 2026, trainee students have been authorised to administer vaccines and injectable medicines during their placement in community pharmacy, provided they are authorised by the placement supervisor and supervised by a professional with active competence.

Objective: To ensure training in AVIM for all MDPC students, thereby increasing the number of pharmacists qualified to provide this service, and to analyse how universities incorporated the SPF training framework into their curricula.

Method: Universities delivering the MDPC were asked to report how the theoretical and practical contents of the PPS training framework had been integrated into the 2025/2026 curriculum, including: the curricular units involved; the methods used to develop competence; the hours allocated to each component; and the integration of basic life support training.

Results: In the 2025/2026 academic year, six of the ten universities had already trained MDPC students in AVIM. As of 18 March 2026, 123 students were undertaking their curricular placement and were able to administer vaccines and injectable medicines under supervision. This number is expected to increase during the final semester of the academic year. From 2026/2027 onwards, all ten universities are expected to provide AVIM training.

Conclusion: All universities were able to integrate the PPS training framework into their curricula, preparing future pharmacists for this role and reducing the need for additional post-graduate training. This approach enables pharmacists to enter professional practice ready to deliver AVIM safely, effectively and to a high standard, while expanding the qualified workforce available to provide this service.

Gut microbial TCDCA promotes blood–brain barrier impairment by activating VSMC apoptosis through TGR5 pathway in Parkinson's disease

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Background: Parkinson's disease (PD) is a prevalent neurodegenerative disorder worldwide, but no disease-modifying therapies are currently available. Emerging evidence has suggested that microbiota-derived metabolites play critical roles in PD through the gut–brain axis. However, the key microbial metabolites driving disease progression remain unclear. Notably, bile acids have recently been implicated as important signalling molecules, but their specific contributions and mechanisms in PD remain poorly defined. We aimed to identify pathogenic bile acids and elucidate their mechanistic role in PD.

Method: We performed plasma metabolomics and faecal metagenomics in a discovery cohort (50 PD and 50 healthy controls) and an independent validation cohort (20 PD and 20 healthy controls). In addition, a rotenone-induced mouse model and a vascular smooth muscle cell (VSMC)-specific TGR5 knockout mouse model were established to evaluate the effects of taurochenodeoxycholic acid (TCDCA) in PD. Specifically, we evaluated motor and gastrointestinal functions, blood-brain barrier (BBB) integrity, neuroinflammation, and tissue pathology. Mechanistically, RNA sequencing, molecular docking, and in vitro assays utilizing MOVAS cells were conducted to validate TCDCA-induced mitochondrial apoptosis.

Results: Multi-omics analyses revealed disrupted bile acid metabolism in PD, with TCDCA markedly elevated and strongly correlated with clinical severity. Metagenomic profiling demonstrated reduced microbial diversity and a significant depletion of bile salt hydrolase (BSH)-encoding bacteria, accompanied by decreased bsh gene abundance, which was inversely correlated with circulating TCDCA levels, indicating impaired microbial deconjugation capacity. In the rotenone-induced mouse model, TCDCA administration significantly aggravated PD-like phenotypes, including motor impairment and gastrointestinal dysfunction. These changes were accompanied by increased systemic and central TCDCA accumulation and exacerbated dopaminergic neurodegeneration. Mechanistically, TCDCA induced profound BBB disruption, as evidenced by decreased tight junction protein expression, increased Evans blue extravasation, and ultrastructural vascular damage. Furthermore, transcriptomic and deconvolution analyses identified VSMCs as a key affected cell population, with reduced expression of canonical markers. Both in vivo and in vitro experiments demonstrated that TCDCA triggered VSMC apoptosis, characterised by caspase-3 activation. Further mechanistic studies showed that TCDCA activates TGR5 signalling in VSMCs, leading to PKCα phosphorylation and mitochondrial apoptotic pathway activation, including cytochrome c release, increased BAX, and decreased Bcl-2 expression. Genetic deletion of TGR5 specifically in VSMCs markedly rescued behavioural deficits, preserved BBB integrity, and attenuated neurodegeneration. Therapeutically, supplementation with BSH-high or Lactobacillus reuteri L. reuteri significantly reduced TCDCA levels across faeces, serum, and brain, improved motor and gastrointestinal function, restored BBB integrity, and suppressed systemic and neuroinflammation. Importantly, these protective effects were abolished in a BSH-deficient strain, confirming that microbial bile acid deconjugation is essential for efficacy.

Conclusion: The integrative mutli-omics analyses of human cohorts identify the conjugated bile acid TCDCA as a previously unrecognized microbial metabolite markedly elevated in PD patients and strongly associated with disease severity. Further experiments validate that TCDCA accumulates in the midbrain, disrupts BBB integrity through TGR5–PKCα–mitochondrial apoptosis in VSMCs, and thereby accelerates dopaminergic neurodegeneration in PD. These findings provide translational and mechanistic evidence that microbial TCDCA is a promising therapeutic target for PD modification that drives VSMC-mediated BBB impairment in the disease pathogenesis.