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Volume Editors Rafal J. Al-Saigh, Argyro Kyriakaki, and Apostolos Zarros

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Details

3rd International Babylon Conference on Clinical and Experimental Pharmacological Research (3rd IBC-CEPR)

Hillah; June 29–30, 2026

Conference organised under the auspices of the Ministry of Higher Education and Scientific Research of the Republic of Iraq, the University of Babylon, Madenat Alelem University, the College of Pharmacy of the University of Babylon, and the College of Pharmacy of Madenat Alelem University

Objectives

> mapping the Iraqi pharmacological research activity and priorities
> facilitating global collaboration

Topics

advances in pharmacological research; antimicrobial resistance and antibiotics; artificial intelligence in pharmacology; challenges in pharmacokinetics; drug delivery; ethnopharmacology; history of drug discovery; new therapeutic trends; pharmaceutical and clinical chemistry; pharmaceutics; pharmacology education; pharmacovigilance and drug safety; physiology and pathophysiology; social pharmacology; trends in nanopharmacology

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PREFATORY NOTE

The 3rd International Babylon Conference on Clinical and Experimental Pharmacological Research: prioritising drug safety, *in silico* drug screening, and the reliability assessment of novel *in vitro* tools for pharmacodynamic simulations

Rafal J. Al-Saigh, Argyro Kyriakaki, and Apostolos Zarros

Received: 27 June 2026; Accepted: 30 June 2026; Published: 01 July 2026

This third iteration of the International Babylon Conference on Clinical and Experimental Pharmacological Research marks a decisive step in the maturation of one of the most important events in Iraq's emerging pharmacological research ecosystem. Building on the foundations laid by the first two conferences (taking place in 2024 and 2025) – each of which had two main objectives, namely the mapping of the Iraqi pharmacological research and priorities, and the facilitation of the establishment of international collaborations in the field [1,2] – this year's conference advances the conversation into domains that are rapidly reshaping global drug development and use. In this third conference, focus shifts toward three interdependent pillars of pharmacological research: drug safety, *in silico* drug screening, and the assessment of novel *in vitro* tools for the undertaking of more reliable pharmacodynamic simulations. Among these pillars, drug safety remains a cornerstone of therapeutic development, and this conference successfully highlights the importance of a systematic evaluation of toxicity, off-target effects, and population-specific risk factors in both Iraq and abroad. The second pillar – *in silico* drug screening – responds directly to the calls for the adoption of innovative methodologies capable of reducing the experimental (and the cost-associated) burden while increasing predictive power. Given that the available computational screening platforms can allow researchers to interrogate vast chemical libraries, model ligand–receptor interactions, and forecast pharmacokinetic behaviour with unprecedented efficiency, their effective integration into the Iraqi research workflows represents both a technological leap as well as a pragmatic response to the current resource constraints. Finally, the third pillar highlighted in this year's conference is the reliability assessment of novel *in vitro* pharmacodynamic tools, and reflects the conference scientific committees' longstanding emphasis on methodological robustness. As the credibility of preclinical experimental findings depends largely on the physiological relevance and the reproducibility of the "models" employed, the rigorous and ongoing (re)validation of the latter becomes increasingly essential and should go hand-in-hand with the pursuit of novelty. This year, the conference takes place at the College of Pharmacy

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of the University of Babylon, in Hillah, Iraq, on the 29th and 30th of June 2026, under the auspices of the Ministry of Higher Education and Scientific Research of the Republic of Iraq, the University of Babylon and its College of Pharmacy, as well as Madenat Alelem University and its College of Pharmacy. The conference proceedings are hereby graciously hosted by *Colloquia Erudita*; a recently established, open-access series of proceedings published by St Cuthbert Press Ltd (Stanley, England, UK). In this volume, our editorial interventions have been minimal while aiming to ensure that the proceedings abstracts maintain the levels of accuracy, clarity, and consistency that have characterized the conference's previous proceedings. To this end, we are grateful to all conference contributors for their collaboration and patience, and we hope that this volume will serve as a testament to the efforts of the conference's steering and scientific committees (led by the Dean of the College of Pharmacy of the University of Babylon, Professor Hussam W. Al-Humadi) to strengthen scientific dialogue and to advance pharmacological research at a local level and in line with international standards.

Keywords

adverse effects; computational screening; *in vitro* methodology; Iraq; pharmacology

Acknowledgements

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Conflicts of interest statement

RJA, AK, and AZ are a professor, a visiting professor, and an honorary professor, respectively, at the University of Babylon. Moreover, RJA and AK serve as series editors of the hosting open-access series (*Colloquia Erudita*), while AZ holds a majority ownership stake in its publisher (St Cuthbert Press Ltd). In order to eliminate any potential conflict-of-interest concerns, the publisher has waived the entirety of the publication fees for this volume of proceedings, ensuring that the University of Babylon bore no financial cost whatsoever for its production or dissemination.

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PROCEEDINGS ABSTRACT

Endocrinopathies associated with immune checkpoint inhibitors: incidence, clinical presentation, and management

Angeliki M. Stamatouli

Received: 01 April 2026; Accepted: 04 April 2026; Published: 05 April 2026

Immune checkpoint inhibitors (CPIs) have fundamentally shifted the oncology landscape by blocking inhibitory pathways that tumours exploit to evade the immune system. By targeting proteins such as the cytotoxic T-lymphocyte associated protein 4 (CTLA-4; e.g., ipilimumab), the programmed cell death protein 1 (PD-1; e.g., pembrolizumab and nivolumab), and the programmed death-ligand 1 (PD-L1; e.g., atezolizumab), these therapies unleash T-cell activity against malignant cells. However, this non-specific immune activation frequently leads to immune-related adverse events (irAEs). Endocrinopathies are among the most common and distinct irAEs, characterised by their often sudden onset and high likelihood of becoming permanent, thereby requiring vigilant pharmacological intervention and lifelong replacement therapy. The mechanisms of the CPI-induced endocrinopathies are distinct from those of traditional autoimmune diseases and vary by drug class. While thyroid dysfunction is the most common endocrinopathy across all CPIs, hypophysitis is the "signature" toxicity of CTLA-4 blockade (e.g., ipilimumab). Hypophysitis is significantly less common with PD-1 / PD-L1 monotherapy. In contrast, anti-PD-1 / PD-L1 therapies are more frequently associated with thyroiditis and type 1 diabetes mellitus (CPI-DM). Combination of anti-CTLA-4 with anti-PD-1 treatments significantly increases the risk of hypophysitis and multi-gland involvement due to a synergistic immune activation at multiple checkpoints. The clinical presentation of hypophysitis is variable, but more commonly includes central adrenal insufficiency. Thyroid dysfunction is the most frequent endocrine irAE. It often manifests as a transient thyrotoxic phase due to destructive thyroiditis, followed by a rapid transition to permanent hypothyroidism. Insulin-deficient diabetes is a rare (~1%) medical emergency, often presenting with severe hyperglycaemia and / or diabetic ketoacidosis. It is characterised by rapid β-cell destruction. Patients usually present with low or undetectable C-peptide levels and, in 30%–50% of the cases, with diabetes auto-antibodies. Finally, primary adrenal insufficiency is rare, but life-threatening; it is distinguished from hypophysitis by the presence of mineralocorticoid deficiency (hyperkalaemia / hyponatraemia). As far as the diagnostic work-up and monitoring are concerned, vigilant screening is mandatory because symptoms like fatigue and nausea are non-specific and can easily be attributed to the underlying cancer. The baseline testing of thyroid-stimulating hormone, free thyroxine, and morning cortisol levels is essential. Throughout treatment, clinicians should monitor thyroid function and blood glucose levels at every infusion cycle. In cases of suspected hypophysitis, MRI may show pituitary enlargement, but treatment should not be delayed for

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imaging if the patient is haemodynamically unstable. A critical distinction in managing endocrine irAEs compared to other organ-specific toxicities (such as colitis or pneumonitis) is the role of immunosuppression. While high-dose glucocorticoids are used to dampen inflammation in other organs, they rarely reverse endocrine gland destruction. Consequently, management shifts from "curing" the inflammation to hormone replacement. A vital pharmacological pearl is the sequence of replacement: adrenal insufficiency must be treated with hydrocortisone before initiating levothyroxine to prevent the precipitation of an acute adrenal crisis due to increased metabolic clearance of cortisol. For CPI-DM, insulin is the only viable treatment; oral hypoglycaemics are ineffective due to the absolute insulin deficiency. In conclusion, as CPI use becomes standard across more cancer types, healthcare providers must recognise that endocrine toxicities are unique "side effects" that persist long after the drug is discontinued. Patient education on the permanent nature of these conditions is paramount for the prevention of mortality while maintaining the oncological benefits of immunotherapy.

Keywords

cancer; endocrinopathies; immune checkpoint inhibitors; immune-related adverse events; toxicities

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Conflicts of interest statement

None to declare.

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PROCEEDINGS ABSTRACT

Evolving role of surgical intervention in spontaneous intracerebral haemorrhage: evidence from the STICH, MISTIE II / III, and ENRICH trials

Alexios Bimpis

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Spontaneous intracerebral haemorrhage (ICH) remains one of the most devastating forms of stroke, as it is associated with high mortality and significant long-term disability, while optimal ICH management strategies continue to be debated. Historically, the surgical evacuation of the haematoma has been widely practiced in ICH cases; however, the landmark Surgical Trial in Intracerebral Haemorrhage (STICH) has demonstrated no overall benefit of early open craniotomy over initial conservative treatment in patients with supratentorial ICH, resulting in a major shift away from surgical intervention in routine clinical practice [1]. Subsequently, attention has shifted toward minimally invasive surgical techniques aiming to reduce the ICH-associated secondary brain injury while minimizing operative morbidity. The Minimally Invasive Surgery with Thrombolysis in Intracerebral Haemorrhage Evacuation (MISTIE) project, including the MISTIE II and MISTIE III trials, has evaluated the effects of catheter-based haematoma evacuation combined with intralesional thrombolysis through the use of recombinant tissue plasminogen activator. While these trials have demonstrated that minimally invasive approaches can achieve a significant haematoma volume reduction and are generally safe, they have failed to demonstrate any statistically significant improvement in terms of functional outcomes at 180 days in the overall patient population, thereby maintaining uncertainty regarding their clinical utility [2]. More recently, the Early Minimally Invasive Removal of Intracerebral Haemorrhage (ENRICH) trial has provided new insights and has demonstrated that the early minimally invasive parafascicular surgical evacuation, particularly when performed within 24 h of the symptoms' onset, is associated with improved functional outcomes (compared to those of the standard medical management) in selected patients with lobar haemorrhages [3]. These findings suggest that the benefit of surgical intervention in ICH is highly dependent on appropriate patient selection, the ICH location, the timing of the intervention, and the specific surgical technique employed. The emerging paradigm indicates a transition from generalized scepticism toward a more refined and individualized surgical strategy in the surgical management of ICH. Although conventional open craniotomy remains of limited value in unselected ICH cases, the early minimally invasive evacuation appears to offer meaningful functional benefits in carefully selected patients. Future studies should prioritize a focus on the integration of advanced imaging biomarkers, the optimization of surgical timing, and the development of standardized selection criteria allowing us to further define the role of surgery in the management of ICH.

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Keywords

ENRICH trial; intracerebral haemorrhage; minimally invasive neurosurgery; MISTIE trials; STICH

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Conflicts of interest statement

None to declare.

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PROCEEDINGS ABSTRACT

The application and therapeutic utility of wearable devices in Parkinson's disease and stroke: current state of evidence

Lisa Hagens and Konstantinos Kalafatakis

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Wearable devices have emerged as promising tools to support objective assessment, monitoring, and rehabilitation in neurological disorders. Parkinson's disease (PD) and stroke, despite their differing pathophysiology, share functional consequences including impaired motor control, gait disturbance, and reduced independence, thereby creating overlapping opportunities for technology-enabled clinical support. Devices range from accelerometers, wristwatches, limb sensors, as well as motion and force sensors to emerging electrochemical platforms. These systems aim at optimising medication and facilitating real-time symptom tracking, remote monitoring, and rehabilitation assistance. We have performed an extensive literature review on the field of the use of wearable devices in PD and stroke; our objectives included consolidating current evidence for PD- and stroke-focused wearable devices, outlining strengths, technical capabilities, clinical practicality, and barriers to real-world deployment. Wearables were surveyed by clinical function and sensing modality. In PD, wearables were predominantly used for the characterization of motor symptoms and treatment response, including accelerometry for tremor, bradykinesia, and motor fluctuation, inertial and force sensors for gait and balance analysis, and emerging biochemical sensing for pharmacological monitoring [1]. The most frequently applied types of wearable devices are accelerometers, motion and force sensors, and electrochemical sensors. Accelerometers are the most extensively researched PD sensors, providing highly specific, real-time symptom tracking (particularly for bradykinesia and motor fluctuations, which correlate closely with dopaminergic levels); thus, they have the potential to reliably monitor treatment efficacy and its chronopharmacological aspects [2]. Similarly, force and motion sensors represent another category of wearable devices frequently utilised for the monitoring of gait and fine hand movements in PD. More recently, early-stage electrochemical sensor-based studies have been designed for the continuous monitoring of the optimal timing and dose of PD medications, by continuously estimating, for example, levodopa levels in the interstitial fluid of PD patients. In stroke, wearables support rehabilitation *via* activity trackers, limb sensors, instrumented footwear, and sensor-assisted orthoses in order to quantify movement, promote repetition, and support home therapy [3]. The most frequently used wearable devices in stroke include shoe insoles, wristwatches, limb sensors, and robotic gloves. Different shoe inserts have been researched in hope of improving the management of gait abnormalities after stroke, assisting rehabilitation, and transmitting kinesiological data to the treating physicians for remote assessment. Wristwatches have also been recruited with the aim to

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increase post-stroke recovery: they monitor baseline activity and walking distance throughout the day as well as during exercises, and remind patients to perform exercises, thereby improving consistency, motivation, and the function of the affected limb. Limb sensors have been employed in studies optimising remote management *via* exercise tracking with features allowing them to monitor muscle activity and gait symmetry aiming at preventing falls. Finally, robotic and smart gloves represent the most extensively studied wearable devices in post-stroke rehabilitation, aiming to improve fine motor control and to restore upper limb function. Wearable devices increasingly integrate digital feedback systems and remote data transmission. While feasible and sensitive to clinically relevant changes, the assessed studies were limited by methodological variabilities, small-cohort sizes, short follow-ups, and inconsistent benchmarking. Wearable technologies provide a shared technological foundation for addressing motor dysfunction in PD and stroke, thereby providing a context-aware assessment. Clinical impact is likely to expand with improved standardisation, robust longitudinal evaluation, and closer alignment with clinical workflows and patient-centred outcomes.

Keywords

individualised therapeutics; Parkinson's disease; rehabilitation; stroke; wearable devices

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Conflicts of interest statement

None to declare.

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PROCEEDINGS ABSTRACT

Biochemical mechanisms and clinical efficacy of glucagon-like peptide-1 receptor agonists in metabolic regulation: a comprehensive review

Rawnaq Kadhim Salih

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The global rise in obesity prevalence has catalysed the development of advanced pharmacological interventions. Amongst these, glucagon-like peptide-1 (GLP-1) receptor agonists have emerged as the result of a critical intersection between metabolic biochemistry and clinical pharmacology. GLP-1 receptor agonists are effective in improving metabolic regulation by enhancing insulin secretion, lowering glycated haemoglobin levels, and promoting weight loss. They also reduce cardiovascular risk, improve lipid and blood pressure profiles, and carry a low risk of hypoglycaemia, making them valuable in managing type 2 diabetes mellitus and metabolic syndrome. They work by mimicking the effects of the incretin hormone GLP-1, which is released from the intestine in response to food intake, thereby enhancing glucose-dependent insulin release, suppressing glucagon release, slowing gastric emptying, and increasing satiety, leading to improved blood glucose control and weight reduction. This review explores the biochemical pathways through which these agents regulate systemic metabolism besides simple appetite suppression. A comprehensive analysis was conducted on current and previous clinical data and biochemical profiles of anti-obesity drugs (GLP-1 receptor agonists) that are designed to be delivered subcutaneously. The focus was on the signalling pathways involved and the role of these drugs in enhancing glucose-dependent insulin secretion and delaying gastric emptying. Clinical data indicate a weight reduction over long-term use of these drugs, accompanied by improved glycaemic control and reduced markers of systemic inflammation. Agents such as semaglutide, liraglutide, and tirzepatide demonstrate significant and sustained weight reduction, representing an effective, minimally invasive option for long-term obesity management. These agents are increasingly being used as primary therapies for obesity, metabolic dysfunction, and established cardiovascular diseases in addition to type 2 diabetes mellitus. Subcutaneously-administered GLP-1 receptor agonists are absorbed into the systemic circulation after injection. In the pancreas, these agents enhance glucose-dependent insulin secretion from β-cells while suppressing glucagon release from α-cells, with indirect inhibition *via* paracrine signalling (somatostatin from δ-cells) leading to improved glycaemic control and reduced hepatic glucose production. In the brain, particularly within the hypothalamus, they activate satiety pathways and reduce hunger-inducing signals, thereby significantly decreasing appetite and food intake. Additionally, these drugs influence reward-related pathways, helping to reduce cravings and hedonic eating. Within the gastrointestinal system, the GLP-1 receptor agonists modulate the release of appetite-related hormones (such as ghrelin and peptide YY)

Salih R. K.: Biochemical mechanisms and clinical efficacy of glucagon-like peptide-1 receptor agonists in metabolic regulation: a comprehensive review. *Colloq. Erud.* 1: 7–8 (2026).
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and delay gastric emptying, thereby prolonging the user's feeling of satiety after meals and reducing postprandial glucose levels. At the molecular level, GLP-1 receptor agonists activate cAMP-dependent signalling pathways that stimulate insulin secretion, promote β-cell survival, modulate ion channel activity, and affect central appetite-regulating neurocircuits; in essence, these agents exert metabolic and anti-obesity effects. They also have the ability to reduce pro-inflammatory cytokine levels (such as those of tumour necrosis factor-alpha and interleukin-6), thereby contributing to improved serum metabolic profiles. The most common side effects associated with GLP-1 receptor agonist use include nausea, vomiting, and injection-site reactions. Important precautions involve the risk of developing acute pancreatitis and the FDA's boxed warning against the use of these drugs by individuals with a personal or family history of medullary thyroid carcinoma and hereditary conditions associated with this type of cancer. Biochemical evidence suggests that these medications do not merely act as anorexiants, but induce significant changes in metabolic rate and adipose tissue thermogenesis. In conclusion, the integration of GLP-1 receptor agonists into obesity management represents a shift toward targeting the underlying biochemical dysfunctions of metabolic syndrome. Future research studies should focus on the long-term metabolic "reprogramming" induced by these agents and their potential in treating non-alcoholic fatty liver disease.

Keywords

biochemistry; GLP-1 receptor agonists; insulin sensitivity; metabolic pathways; obesity

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COLLOQUIA ERUDITA 2026; 1: 9–10

PROCEEDINGS ABSTRACT

Pharmacological approach to tooth regeneration *via* uterine sensitization-associated gene-1 inhibition

Zahraa Abbas Mohsin, Mohammed Obies, and Aymen Bash

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Oral diseases remain a major global public health burden. Although oral healthcare has improved in many regions, common conditions such as dental caries and periodontal disease continue to be highly prevalent. These disorders frequently result in tooth loss and negatively impact overall health, quality of life, and well-being. Current treatments such as dental implants are widely considered as standard treatment for the replacement of missing teeth. However, they do not actually restore a real biological tooth and they are associated with several complications; amongst these, peri-implantitis is the most significant and is characterized by an inflammatory destruction of peri-implant tissues and progressive bone loss, ultimately leading to implant failure. These limitations highlight the need for effective pharmacological strategies that promote true biological tooth regeneration [1]. Recent studies have focused on a protein called "uterine sensitization–associated gene-1" (USAG-1), which is known to suppress tooth development by blocking signalling pathways such as those of the bone morphogenetic protein (BMP) and Wnt [2]. When USAG-1 was inhibited in animal studies, *de novo* tooth formation was observed. From a pharmacological perspective, USAG-1 represents a potential therapeutic target for the induction of tooth regeneration. Experimental approaches such as monoclonal antibody therapy (widely used in cancer therapy to target specific proteins involved in disease progression) and RNA-based gene silencing have been shown to decrease USAG-1 activity, thereby restoring Wnt and BMP signalling and reactivating the dormant odontogenic potential [3]. Similarly to other vertebrate organs, tooth development (odontogenesis) is known to be regulated by reciprocal interactions between epithelial and underlying mesenchymal tissues through multiple signalling pathways, including the Wnt and BMP ones. More specifically, the Wnt signalling is activated at the earliest stage of odontogenesis and is responsible for initiating tooth formation by activating odontogenic stem cells and promoting the formation of the tooth bud through increased cellular proliferation. In contrast, the BMP signalling becomes more prominent during later stages, where it regulates cellular differentiation and guides the formation of specialized dental cells (such as ameloblasts and odontoblasts) responsible for enamel and dentin formation. Although USAG-1-targeting therapies are not yet clinically available, they represent a promising area of pharmaceutical research, in which pharmacists play a key role in drug development, safety evaluation, and future clinical application(s). However, several limitations must be considered. Most current evidence is based only on animal models; therefore, the inhibition of USAG-1 in humans remains uncertain. In addition, the long-term safety of monoclonal antibody or RNA-based therapies also requires further investigation, particularly in light of the known adverse effects associated with biologic therapies. Moreover, RNA-based approaches face important

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challenges related to effective drug delivery and optimal dosing strategies. Therefore, additional preclinical and clinical investigations are required in order to assess the safety, efficacy, and feasibility of USAG-1 inhibition before any relevant therapeutic strategies can be successfully introduced into clinical practice.

Keywords

BMP; drug development; oral diseases; tooth regeneration; USAG-1

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Conflicts of interest statement

None to declare.

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COLLOQUIA ERUDITA 2026; 1: 11–12

PROCEEDINGS ABSTRACT

Effects of dietary probiotic supplementation on the serum reproductive hormone levels of rats subjected to high-fat diet- and letrozole-induced experimental polycystic ovary syndrome

Bassam Hasan, Aymen A. Bash, and Lena Fadhil Hamza

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The polycystic ovary syndrome (PCOS) is a complicated endocrine disease that occurs in women of reproductive age and that is marked by hyperandrogenism, ovulation disorders, and multiple ovarian cysts. Other related symptoms include insulin resistance, hirsutism, and obesity, while the biguanide metformin is the most appropriate drug for the treatment of PCOS. Intestinal microbiota plays a role in maintaining metabolic and hormonal health, while dysbiosis disrupts the regulation of reproductive hormones, leading to increased intestinal permeability, intestinal microbiota imbalance, and – finally – to PCOS development. This aim of this study was to examine the impact of probiotic supplementation (mostly of *Lactobacillus* and *Bifidobacterium*) on the serum levels of testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) in female rats undergoing an experimental simulation of PCOS with the use of high-fat diet (HFD) and letrozole. To this end, 28 female Wistar rats were randomly assigned into four groups (n=7 each): a control group, a PCOS group, a metformin-treated PCOS group, and a probiotic-receiving PCOS group. The HFD that was used in this study was a commercial product obtained from Research Diets, Inc. (USA) with a total kcal value of 4.73 kcal/g; this HFD was coupled with letrozole (given orally, *via* gavage, at a dose of 1 mg/kg/day) in order to induce PCOS. After 21 days of stimulation, the PCOS rats were returned to a normal diet (like the one fed to the control group). Metformin was then administered at a dose of 300 mg/kg/day in distilled water, while probiotic supplementation with a combination of *Bifidobacterium* and *Lactobacillus* bacteria (using a commercial product obtained from Medwise Healthcare Limited, UK) took place at a dose of 1×10^9 CFU/day for 28 days. Daily vaginal swabs were obtained throughout the induction period in order to determine the oestrous cycle irregularity and to confirm PCOS. Subsequently, cardiac puncture was used in rats anesthetized with diethyl ether in order to obtain blood samples, and serum hormone levels were measured with ELISA. Rat serum testosterone and LH levels were found to be significantly increased by 72.11% (p=0.003) and 89.25% (p=0.001), respectively, in the PCOS group, while the serum FSH levels were found to be significantly decreased by 38.02% (p<0.001) in the same group when compared to those of the control group. The metformin-treated PCOS group exhibited a decrease by 35.09% (p>0.05) in its serum LH levels when compared

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to those of the PCOS group. Finally, the herein employed probiotic supplementation led to a moderate reduction of the serum testosterone and LH levels by 15.31% (p>0.05) and 23.73% (p>0.05), respectively, when compared to those of the PCOS group. The serum FSH levels were found to be significantly lower (by 30.67%; p=0.004) in the probiotic-receiving PCOS group when compared to those of the control group. Our findings indicate that despite the fact that probiotic supplementation has an impact on the reproductive axis, it cannot be used as the only intervention for the restoration of hormonal balance in female rats undergoing an experimental simulation of PCOS.

Keywords

letrozole; metformin; PCOS; probiotics; testosterone

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Conflicts of interest statement

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COLLOQUIA ERUDITA 2026; 1: 13–14

PROCEEDINGS ABSTRACT

Effects of telmisartan and of a common dandelion (*Taraxacum officinale*) extract on the serum reproductive hormone levels of rats subjected to high-fat diet- and letrozole-induced experimental polycystic ovary syndrome

Zainab Hafedh Alsharifi and Aymen A. Bash

Received: 12 March 2026; Revised: 20 May 2026; Accepted: 14 June 2026; Published: 15 June 2026

Polycystic ovary syndrome (PCOS) is a common endocrine disorder of reproductive-age women, characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology, and causing a range of reproductive, metabolic, dermatological, and psychological issues. The aetiology of PCOS involves a complex interplay of environmental, genetic, and lifestyle factors, where a family history can elevate the risk; although the complete pathophysiology remains unclear, hyperandrogenism, insulin resistance, inflammation, and oxidative stress are considered significant contributors. Our study's aim was to investigate the effects of telmisartan and of a *Taraxacum officinale* (common dandelion) extract on a rat model of PCOS, by measuring the serum levels of testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH). A total of 35 female Wistar rats were randomly divided into five groups (n=7 each): a control group, a PCOS group, a metformin-treated PCOS group, a telmisartan-treated PCOS group, and a *Taraxacum officinale* extract-treated PCOS group. PCOS was induced through high-fat diet (HFD) and letrozole administration. The HFD that was used in this study was a commercial product obtained from Research Diets, Inc. (USA) with a total kcal value of 4.73 kcal/g, and it was coupled with letrozole (given orally, *via* gavage, at a dose of 1 mg/kg/day) in order to induce PCOS. After 30 days of stimulation, the PCOS rats were returned to a normal diet (like the one fed to the control group). Metformin was then administered at a dose of 300 mg/kg/day, telmisartan at a dose of 10 mg/kg/day, and the *Taraxacum officinale* extract at a dose of 500 mg/kg/day (*via* oral gavage) for 28 days. Vaginal swabs were obtained daily and throughout the induction period in order to determine the oestrous cycle irregularity and to confirm PCOS. Subsequently, cardiac puncture was used in rats anesthetized with diethyl ether in order to obtain blood samples, and serum hormone levels were measured with ELISA. The administration of HFD and letrozole significantly increased the serum testosterone and LH levels by 191.53% (p=0.001) and 85.2% (p=0.021), respectively, while significantly reducing the serum FSH levels by 50.1% (p=0.001) when compared to those of the control group. On the other hand, the metformin-treated PCOS

Alsharifi Z. H., Bash A. A.: Effects of telmisartan and of a common dandelion (*Taraxacum officinale*) extract on the serum reproductive hormone levels of rats subjected to high-fat diet- and letrozole-induced experimental polycystic ovary syndrome. *Colloq. Erud.* 1: 13–14 (2026).
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group exhibited a reduction in its serum testosterone and in its LH levels by 50.85% (p=0.012) and 35.86% (p=0.136), respectively, and a 52.77% increase (p=0.215) in its serum FSH levels when compared to those of the PCOS group. The telmisartan-treated PCOS group exhibited a significant reduction in its serum testosterone levels by 61.72% (p=0.001), a reduction in its serum LH levels by 32.18% (p=0.254), and an increase in its serum FSH levels by 40.11% (p=0.751) as compared to those of the PCOS group. Finally, the *Taraxacum officinale* extract-treated PCOS group exhibited a reduction in its serum testosterone and LH levels by 13.1% (p>0.05) and 21.66% (p>0.05), respectively, while its serum FSH levels were found increased by 27.76% (p>0.05) when compared to those of the PCOS group. Overall, the administration of telmisartan and of the *Taraxacum officinale* extract reduced the serum LH and increased the FSH levels, thereby suggesting a potential regulation of the gonadotropin signalling; the same treatments also managed to reduce the serum testosterone levels, thereby suggesting an ability to modulate androgen synthesis.

Keywords

metformin; PCOS; reproductive hormones; *Taraxacum officinale*; telmisartan

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COLLOQUIA ERUDITA 2026; 1: 15–16

PROCEEDINGS ABSTRACT

Development and physicochemical characterization of selenium nanoparticles stabilized and functionalized by hyaluronic and gallic acid

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The discovery of targeted, biocompatible nanocarriers represents a substantial transformation in the field of cancer therapeutics by addressing critical limitations of conventional chemotherapy (including nonspecific toxicity, unfavourable pharmacokinetics, and the emergence of multidrug resistance). Nanotechnology-based drug delivery systems have emerged as effective methods of addressing these limitations. Selenium (Se) nanoparticles (SeNPs), in particular, have garnered considerable interest due to their unique physicochemical and biological characteristics, including low toxicity, biocompatibility, antioxidant activity, and prospective medicinal utility. The surface modification and functionalization of SeNPs with biocompatible compounds can enhance their stability, targeting efficacy, and overall performance as nanocarriers. This study has focused on varying hyaluronic acid (HA) to Se and gallic acid (GA) to Se mass ratios in order to identify the optimal formulation for generating SeNPs with desirable particle size distribution, high homogeneity, and adequate colloidal stability for nanodrug delivery applications. We have effectively produced and physiochemically characterized SeNPs that are stabilized and functionalized with HA and GA (HA/GA-SeNPs). HA was chosen for its superior biocompatibility and its capacity to improve nanoparticle stability and cellular targeting, while GA was used as a natural polyphenolic stabilizer and functionalizing agent. The nanoparticles were produced using sodium selenite (as the Se precursor) and ascorbic acid (as the reducing agent) under regulated conditions in the presence of HA and GA. HA solutions (5 mg/mL) were subsequently added in varying concentrations in order to produce several formulations with HA to Se ratios of 5:1, 7.5:1, 10:1, and 12.5:1. The reaction mixture's obvious colour change from colourless to reddish-orange was the first indication that the nanoparticles had been formed, while the produced HA/GA-SeNPs were characterized by a variety of techniques, including dynamic light scattering and zeta potential assessment. Our results indicate that alterations in the HA to Se and the GA to Se ratios can greatly influence nanoparticle size, surface charge, and dispersity. The examined HA-SeNPs, with HA to Se ratios of 5:1, 7.5:1, 10:1, and 12.5:1, have yielded nanoparticles exhibiting Z-average diameters between 123.6 and 130.8 nm, with polydispersity index (PDI) values ranging from 0.085 to 0.182. Zeta potential tests varied from -12.8 to -24.8 mV, thereby signifying adequate colloidal stability. The HA to Se ratio of 7.5:1 exhibited optimal particle homogeneity, as evidenced by a PDI value of 0.085. Further optimization was conducted by integrating GA at various GA to Se ratios (0:1, 2.5:1, and 5:1). Our findings indicate that the formulation created at HA to Se and GA to Se ratios of 5:1 bears the most ad-

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vantageous physicochemical characteristics, featuring a Z-average diameter of 98.91 nm, a low PDI value of 0.065, and a zeta potential of -16.7 mV. The results demonstrate the effective synthesis of highly uniform and stable HA/GA-SeNPs of suitable nanoscale dimensions, that could exert enhanced permeability and retention-mediated tumour targeting. In conclusion, the HA/GA-SeNPs synthesized in our study constitute a viable nanoscale platform with excellent physicochemical characteristics and a promising potential as an efficient and selective nanodrug delivery system for anticancer applications.

Keywords

gallic acid; hyaluronic acid; selectivity; selenium; targetability

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COLLOQUIA ERUDITA 2026; 1: 17–18

PROCEEDINGS ABSTRACT

Study of a novel series of 1,2,4-triazole derivatives: synthesis, spectroscopic characterization, and *in silico* assessment as antifungal substances

Saja Alsalhy and Hussam W. Al-Humadi

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Invasive fungal infections pose a major global health issue, especially those arising from drug-resistant *Candida* species, highlighting the need for an ongoing creation of new antifungal medications. This study focuses on the development, formulation, and computational analysis of a novel series of eight 1,2,4-triazole derivatives (D1, D2, E–J) as potential antifungal agents targeting cytochrome P450 14α-demethylase (CYP51). The synthetic method utilized a multi-stage process that started with the Fischer esterification of a derivative of a carboxylic acid and ethanol, resulting in the formation of the corresponding ester. Next, hydrazide was produced by reacting with hydrazine hydrate. Following nucleophilic addition with aryl isothiocyanate produced intermediates (C1 and C2), while the base-promoted cyclization of these intermediates yielded the core 1,2,4-triazole-5(4H)-thione frameworks (D1 and D2). The last step consisted of alkylating the thione's sulfur atom with a range of alkylating agents such as iodoethane, phenacyl bromide derivatives, butyl iodide, and ethyl bromo acetate, yielding a varied collection of target compounds (E–J). All synthesized compounds were analysed and structurally verified through Fourier transform infrared (FTIR), proton nuclear magnetic resonance (^{1}H-NMR), and carbon-13 nuclear magnetic resonance (^{13}C-NMR) spectroscopy. FTIR spectroscopy demonstrated the disappearance of the carbonyl band after cyclization and the forming of compounds D1 and D2, the appearance of C=N stretching around 1608 cm^{-1}, a distinct carbonyl absorption at 1703 cm^{-1} for compound F, and ester-related peaks at 1663 cm^{-1} and 1259 cm^{-1} in the case of compound I. The spectroscopic data validated the successful creation of all intermediates and final products by displaying characteristic absorption bands and chemical shifts that align with the suggested structures. Molecular docking analyses on the CYP51 enzyme (a recognized target for azole antifungal drugs) were also performed through the use of the Molecular Operating Environment software. The docking analysis revealed several of the synthesized compounds to be demonstrating encouraging binding affinities that were similar to those of the reference drug (fluconazole). Throughout the series, the binding energies were advantageous, with two compounds exhibiting the most favourable ones: (i) compound F and (ii) compound I. An in-depth examination of the protein–ligand interactions revealed that the lead compound can establish several hydrogen bonds with active site residues such as threonine and serine, along with many hydrophobic interactions with alanine, leucine, proline, tyrosine, isoleucine, valine, lysine, and cysteine residues. A different promising compound can form an essential hydrogen bond with a cysteine residue, along with hydrophobic interactions and a pi–

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sulphur interaction involving phenylalanine. These interaction patterns suggest a consistent binding within the enzyme's active site. Subsequently, the compounds' ADME (absorption, distribution, metabolism, and excretion) characteristics were forecasted in order to evaluate their drug-likeness. The majority of the derivatives exhibited advantageous physicochemical characteristics, such as suitable molecular weights, counts of hydrogen bond donors and acceptors, and topological polar surface areas within acceptable limits. Most of the compounds exhibited significant gastrointestinal absorption and positive bioavailability ratings, but none of them demonstrated an ability to cross the blood-brain barrier. The majority of compounds adhered to standard drug-likeness criteria, while one of the compounds deviated because of its elevated molecular weight. Consensus log P values reflected significant lipophilicity for all compounds, thereby indicating favourable membrane permeability and potential for tissue distribution.

Keywords

antifungal agents; CYP51 inhibition; molecular docking; synthesis; 1,2,4-triazole

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COLLOQUIA ERUDITA 2026; 1: 19–20

PROCEEDINGS ABSTRACT

A novel polyethylene glycol / graphene oxide composite as a nanocarrier of methylprednisolone: synthesis and physicochemical characterization

Suhad H. Alzubaidy and Shafq Al-Azzawi

Received: 17 March 2026; Revised: 23 May 2026; Accepted: 14 June 2026; Published: 18 June 2026

A popular corticosteroid with strong anti-inflammatory and immune suppressive qualities, methylprednisolone (MP), has a comparatively low cellular absorption and a restricted water solubility that limit its therapeutic efficacy and require a greater dosage, thereby raising the possibility of unfavourable systemic side effects. Therefore, it is crucial to create a drug delivery system that can enhance MP's solubility, stability and targeted distribution; carrier-based nanocarriers have been shown to overcome these restrictions. Because of its enormous surface area, its abundance of oxygen-containing functional groups, and its exceptional aptitude for drug adsorption through pi–pi stacking and hydrogen bonding interactions, graphene oxide (GO) has garnered great attention amongst nanomaterials. GO is a flexible platform for medication-loading and delivery applications. The PEGylation (or surface functionalization with polyethylene glycol; PEG) has been used in order to enhance the stability, dispersibility, and biocompatibility of nanomaterials for various medical applications. We herein report the development of a PEGylated GO nanoparticle as a novel nanocarrier system for the transportation of MP and the enhancement of its delivery. A solution blending method was used in order to synthesize the nanocomposite *via* the dispersion of GO, first ultrasonically in the deionized water and then through the functionalization of its surface so as to improve its stability and compatibility. PEG was then added with constant stirring in order to encourage the interaction between GO and PEG. The mixture was purified using ultracentrifugation so as to remove any unbounded components and to produce stable GO–PEG nanoparticles. MP was loaded onto the PEGylated GO nanocarrier through an adsorption procedure under carefully regulated heating and stirring conditions. Multiple binding sites were made possible by the GO's vast surface area and functional groups, which allow the drug molecules on the nanocarrier matrix to interact effectively. Several approaches were employed in order to clarify the nanocomposite's physicochemical properties and to verify that the GO was successfully functionalized with PEG. In order to detect the interaction between the carrier and the loaded medication (MP), we employed Fourier transform infrared (FTIR) spectroscopy. The structural features and crystalline properties of the developed nanomaterial were examined using X-ray diffraction (XRD) analysis, while energy dispersive spectroscopy (EDS) was used in order to ascertain the elemental composition of the produced nanocomposite. Furthermore, scanning electron microscopy (SEM) revealed details about the surface shape and particle size distribution of the produced nanoparticles. In brief, the GO–PEG nanocomposite was successfully formed, and MP was found to be ef-

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fectively loaded on it. Successful surface functionalization was confirmed through FTIR, displaying distinctive absorption bands related to GO and peaks linked to PEG, as well as interactions between the MP and the GO–PEG matrix. The undertaken XRD analysis revealed structural changes indicative of the creation of a stable structure. Good dispersion was demonstrated by the obtained SEM micrographs, with the latter revealing nanoscale sheets-like structures with comparatively homogeneous shapes. EDS demonstrated a successful drug integration into the GO-based carrier. Our findings suggest that the PEGylated GO could serve as a promising nanocarrier for MP administration due to its advantageous properties and drug-carrier interactions.

Keywords

drug delivery system; graphene oxide; methylprednisolone; PEG; PEGylated graphene oxide

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COLLOQUIA ERUDITA 2026; 1: 21–22

PROCEEDINGS ABSTRACT

Comparative assessment of the effects of resatorvid and sulfasalazine on disease severity, intestinal histopathology, as well as inflammation and oxidative stress markers in a murine experimental model of ulcerative colitis

Baneen Abbas Altaay and Fatima Adnan Alzubaidi

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Ulcerative colitis (UC) is a chronic relapsing inflammatory disease with an incompletely understood aetiology and no definitive cure, in which toll-like receptor 4 (TLR4)-mediated inflammation plays a key pathogenic role and represents a promising therapeutic target. This study aimed at evaluating the protective effects of resatorvid compared with those of sulfasalazine in a dextran sulfate sodium (DSS)-induced experimental model of UC in mice. To this end, 50 adult male BALB/C mice (aged 6–10 weeks; weighing 22–30 g) were randomly assigned into five experimental groups: (i) a control group, (ii) a DSS-induced UC group (in which DSS was administered orally through a cage feeding bottle at a concentration of 5% for 10 days), (iii) a group that has been exposed to DSS and has also received resatorvid (at 3 mg/kg, once daily, intraperitoneally) for 10 days, (iv) a group that has been exposed to DSS and has also received sulfasalazine (at 100 mg/kg, orally, by gavage once, daily) for 10 days, and (v) a vehicle control group that has been exposed to DSS and has also received 1% DMSO (in normal saline, once daily, intraperitoneally) for 10 days. On day 11 of the study, the mice were sacrificed, and colonic tissue was collected for biochemical and histopathological analysis. The levels of the oxidative stress markers myeloperoxidase (MPO) and isoprostane-8, of TLR4, and of the pro-inflammatory cytokines tumour necrosis factor alpha (TNF-α) and interleukin-1 beta (IL-1β) were quantified in the colonic tissues using ELISA kits. Histological examination was performed in order to assess the extent of the mucosal damage and inflammatory cell infiltration. DSS administration induced severe experimental colitis, as evidenced by a significant elevation of the disease activity index (DAI), macroscopic and histopathological scores, oxidative stress markers, and pro-inflammatory cytokine levels, as compared to those of the control group (p<0.001). The tissue levels of TNF-α, IL-1β, MPO, isoprostane-8, and TLR4 were found to be increased by approximately 274%, 262%, 136%, 135%, and 118.3%, respectively, in the DSS-induced UC model mice when compared to those of the control group. No significant differences were observed in the herein assessed parameters between the DSS-induced UC model group and the vehicle- and DSS-treated group. The treatment

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with sulfasalazine significantly reduced the DAI and the macroscopic and histopathological scores, as well as the colonic tissue TNF-α, IL-1β, MPO, isoprostane-8, and TLR4 levels by approximately 51%, 49%, 56%, 44%, 44%, 37%, 34%, and 25%, respectively, when compared with those of the UC model mice (p<0.001). Similarly, the administration of resatorvid was found to significantly attenuate the DSS-induced colonic injury by reducing the DAI, the macroscopic and histopathological scores, and the tissue levels of TNF-α, IL-1β, MPO, isoprostane-8, and TLR4 by approximately 47%, 42%, 47%, 40%, 38%, 35%, 32%, and 44%, respectively, compared with those of the DSS-induced UC model mice (p<0.001). No significant differences were observed amongst the herein assessed parameters between the resatorvid- and the sulfasalazine-treated groups, with the exception of resatorvid demonstrating a significantly greater suppression of the colonic TLR4 expression compared to sulfasalazine (p<0.001). Our findings are supportive of selective TLR4 inhibition as a promising therapeutic strategy for the treatment of UC.

Keywords

oxidative stress; sulfasalazine; TAK-242; TLR4; ulcerative colitis

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Conflicts of interest statement

None to declare.

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COLLOQUIA ERUDITA 2026; 1: 23–24

PROCEEDINGS ABSTRACT

Combining vancomycin with caspofungin against a *Staphylococcus aureus–Candida albicans* coinfection in an *in vitro* pharmacokinetics / pharmacodynamics model: robustness of the modelling and the impact of albumin and platelets

Asmaa S. Dhaidan and Hussam W. Al-Humadi

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Polymicrobial infections involving *Staphylococcus aureus* and *Candida albicans* represent a significant clinical challenge due to their synergistic pathogenicity, enhanced resistance, and high associated mortality rates. These infections are particularly prevalent in immunocompromised patients, and are frequently associated with biofilm-related conditions and bloodstream infections, thereby contributing to increased virulence and reduced susceptibility to antimicrobial agents [1]. Consistent with these concerns, the present study was designed with the aim to evaluate the pharmacodynamic behaviours of vancomycin and caspofungin in combination, through a dynamic *in vitro* pharmacokinetics / pharmacodynamics (PK/PD) model that simulates clinically relevant human drug exposure while incorporating key host-related factors, such as albumin and platelets. To ensure alignment with physiologically relevant conditions, standard reference strains (*S. aureus* ATCC 25923 and *C. albicans* ATCC 90028) were exposed to simulated peak plasma concentrations of vancomycin (3 and 5 mg/L) and caspofungin (10 and 20 mg/L), administered in combination, as well as in the presence of 2% human albumin and 2% activated platelets, either alone or in combination, so as to better replicate *in vivo* environments and to assess the impact of protein binding and of the host immune components on antimicrobial activity. The PK/PD model employed a peristaltic pump system in order to simulate the drug elimination kinetics, achieving a half-life of approximately 24 h and closely mimicking human pharmacokinetic profiles [2]. Pharmacodynamic responses were monitored through serial measurements of optical density at 600 nm, which were also standardized against colony-forming unit counts so as to ensure accuracy and reproducibility. Data were subsequently analysed using the E_{max} model in order to characterize dose–response relationships and to quantify the extent of the occurring antimicrobial activity under different experimental conditions [2]. The undertaken pharmacokinetic simulations demonstrated high reproducibility, with drug concentration time profiles closely matching those of the expected human plasma kinetics and exhibiting minimal inter-experimental variability. Furthermore, the robustness of the pharmacodynamic modelling was support-

Dhaidan A. S., Al-Humadi H. W.: Combining vancomycin with caspofungin against a *Staphylococcus aureus–Candida albicans* coinfection in an *in vitro* pharmacokinetics / pharmacodynamics model: robustness of the modelling and the impact of albumin and platelets. *Colloq. Erud.* 1: 23–24 (2026).
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ed by strong correlation coefficients (R^2 values), thereby confirming the reliability and predictive value of the employed system. The results demonstrated that a combination therapy with vancomycin and caspofungin can produce significantly enhanced antimicrobial effects, particularly in co-infection settings where microbial interactions typically compromise the treatment efficacy. Our findings suggest that a combined administration can led to improved suppression of both bacterial and fungal growth. Notably, the presence of host factors had a measurable impact on drug performance. The inclusion of albumin is particularly relevant given its known effect on reducing the free, pharmacologically-active fraction of highly protein-bound drugs, while activated platelets contribute to host defence through the release of antimicrobial peptides and the modulation of microbial growth dynamics through platelet-mediated immune mechanisms. Interestingly, the combined presence of albumin and platelets produced intermediate effects, thereby indicating a complex interplay between drug binding and host defence factors. In conclusion, this study highlights the importance of integrating host physiological components into *in vitro* PK/PD models so as to achieve a more accurate representation of the *in vivo* conditions. Furthermore, the herein demonstrated impact of albumin on drug activity emphasizes the need to consider protein-binding effects when interpreting *in vitro* results and translating them into clinical practice [3].

Keywords

antimicrobial synergy; caspofungin; pharmacokinetic / pharmacodynamic model; polymicrobial infection; vancomycin

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Conflicts of interest statement

None to declare.

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COLLOQUIA ERUDITA 2026; 1: 25–26

PROCEEDINGS ABSTRACT

Association of age, anthropometric measurements, reproductive history, and critical serum hormone levels with *in vitro* fertilization outcomes in Iraqi women

Rafal H. Jawad and Dhafir Masheta

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Infertility represents a major reproductive health problem, while assisted reproductive technologies, particularly *in vitro* fertilization (IVF), are widely used as effective therapeutic strategies for its management. The present study aimed at investigating the association between age, anthropometric measurements, and serum levels of key reproductive hormone with IVF outcomes among Iraqi women undergoing assisted reproductive treatment. This comparative cross-sectional study included 101 women undergoing IVF treatment. Participants were classified into two groups according to IVF outcome: a successful IVF group (N=60) and a failed IVF group (N=41). Women between 18 and 40 years of age with complete clinical and laboratory records were included in the study. Participants with diagnosed thyroid disease requiring treatment, diabetes mellitus, hyperprolactinaemia, or incomplete laboratory data were excluded from our study in order to reduce potential confounding variables. Anthropometric measurements, including body mass index (BMI), height, and weight, as well as reproductive history (such as gravidity) were also recorded. Blood samples were collected from all participants at the Tiba IVF Center (Hillah, Iraq) between November 2025 and March 2026, and were used in order to measure in them the serum levels of the anti-Müllerian (AMH), follicle-stimulating (FSH), luteinizing (LH), and thyroid-stimulating (TSH) hormones as well as those of oestradiol and prolactin. Hormonal analyses were performed using the VIDAS® Blue automated immunoassay system. Our results revealed maternal age to be significantly higher in the failed IVF group than in the successful IVF group (30.33 *versus* 28.19 years, p=0.028). The age distribution analysis further indicated that IVF success was more common among younger women, while the likelihood of IVF failure increased progressively with advancing maternal age. The anthropometric measurements (including BMI, height, and weight) did not differ significantly between successful and failed IVF groups. Gravidity was also significantly higher among women who achieved successful IVF as compared to those of the women who did not (1.75 *versus* 0.91, p=0.0003). Serum prolactin levels were found to be significantly higher in the women with successful IVF outcomes when compared with those of the women with failed IVF outcomes (25.90 *versus* 18.90 ng/mL, p=0.0002). Similarly, serum TSH levels were significantly higher in the successful IVF group than in the failed IVF group (2.80 *versus* 2.25 μIU/mL, p=0.0228). In contrast, no statistically significant differences were observed between the two groups in terms of their serum levels of AMH, FSH, LH, or oestradiol. Moreover, a correlation analysis revealed a significant positive correlation between AMH and LH levels (r=0.299, p=0.0025) and a signifi-

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cant negative correlation between AMH and FSH levels (r=-0.291, p=0.0033). In conclusion, our findings indicate that age, serum prolactin levels, serum TSH levels, and gravidity are significantly associated with IVF success in Iraqi women, whereas the serum levels of AMH, FSH, LH, and oestradiol, as well as the herein assessed anthropometric measurements were not predictive of the IVF outcome.

Keywords

in vitro fertilization; Iraq; prolactin; reproductive hormones; thyroid-stimulating hormone

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Conflicts of interest statement

None to declare.

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COLLOQUIA ERUDITA 2026; 1: 27–28

PROCEEDINGS ABSTRACT

Development and characterization of a novel nanocomposite hydrogel using polyvinyl alcohol, chitosan, and SiO_2: promising findings for its employment in biomedical applications

Karrar Najeh Chfat, Noor Hadi Aysa, and Shafq Al-Azzawi

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In the contemporary pharmaceutical sciences and nanotechnology, the development of smart drug delivery systems remains a pivotal challenge. Our study represents a comprehensive investigation into the fabrication and characterization of advanced nanocomposite hydrogels (NCHGs) that are specifically engineered so as to optimize structural integrity and morphological performance for drug-loading and various medical applications. The study focuses on a hybrid polymeric architecture primarily synthesized from polyvinyl alcohol (PVA) and chitosan (CS). These polymers were chosen for their inherent biocompatibility and their functional adaptability; properties further enhanced through the strategic incorporation of silica (silicon dioxide; SiO_2) nanoparticles. The objective of this formulation was to create a robust scaffold through a controlled blending and chemical crosslinking, ensuring a highly uniform dispersion of SiO_2 within the network so as to gain mechanical stability and to prevent a premature degradation at specific environments (such as tumor tissues). Hydrogels are widely recognised for their high-water absorption capacity, biocompatibility, and structural similarity to biological tissues, making them suitable for different biomedical applications (such as wound healing, tissue engineering, and drug delivery). The chemical architecture and the successful integration of the components were rigorously validated using Fourier transform infrared spectroscopy. The analysis revealed distinctive characteristic hydroxyl (-OH) and amine ($-NH_2$) groups originating from the PVA/CS matrix, which exhibited significant molecular interaction with the siloxane (Si-O-Si) networks of the SiO_2 nanoparticles. Specifically, the broad band at 3420 cm^{-1} is linked to the overlapping O–H and N–H stretching vibrations of CS, thereby reflecting robust H-bonding in the hydrogel structure. These interactions enhance stability, providing a crosslinked environment that facilitates the loading and retention of therapeutic agents. Furthermore, the structural phase of the composite was examined through X-ray diffraction patterns. The results confirmed that the SiO_2 nanoparticles maintained a purely amorphous state, as evidenced by a broad angle. Conversely, the hydrogel matrix demonstrated a semi-crystalline nature, with distinct crystalline peaks corresponding to PVA regions. The coexistence of these two phases (amorphous SiO_2 and semi-crystalline polymer) proves that the nanoparticles were successfully intercalated into the scaffold without disrupting the

Chfat K. N., Aysa N. H., Al-Azzawi S.: Development and characterization of a novel nanocomposite hydrogel using polyvinyl alcohol, chitosan, and SiO_2: promising findings for its employment in biomedical applications. *Colloq. Erud.* 1: 27–28 (2026).
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overall structural continuity. Surface morphology and internal architecture were further elucidated using scanning electron microscopy; the latter revealed a rugged, highly porous polymeric framework, which acts as a sophisticated host for the nanoparticles. The SiO_2 nanoparticles were observed as bright, spherical entities distributed across the hydrogel surface. Other significant regions demonstrated a highly monodisperse and uniform distribution, while the hydrogel exhibited high mechanical strength (3.76 MPa) and remarkable elasticity (338% elongation). The observed swelling exhibited a controlled behaviour: it increased at acidic pH media and retained its stability in neutral environments with controlled swelling over 48 h, so as to ensure sustained drug release. By controlling the spatial arrangement of these particles, the system is capable of facilitating both a sustained and a regulated release of drugs, thereby improving therapeutic efficacy, ensuring that the drug concentration remains within the therapeutic window for an extended period of time (as in a tumour environment), and significantly reducing systemic toxicity. Overall, the produced NCHG displays significant potential to be applied in different biomedical applications.

Keywords

chitosan; hydrogel; nanocomposite; silicon dioxide; swelling capacity

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COLLOQUIA ERUDITA 2026; 1: 29–30

PROCEEDINGS ABSTRACT

Pioglitazone improves the motor function and attenuates the central and peripheral alpha-synuclein burden in a rotenone-induced model of Parkinson's disease in rats

Ghufran Sapri Abbood, Fatima Adnan Alzubaidi, and Mazin J. Mousa

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Parkinson's disease (PD) is a chronic, progressive neurodegenerative disorder characterized by the selective degeneration of dopaminergic neurons in the substantia nigra pars compacta and the widespread accumulation of alpha-synuclein (α-Syn); an intracellular aggregation-prone protein. The resulting dopamine deficiency in the basal ganglia gives rise to the hallmark motor features of PD, including resting tremor, bradykinesia, gait disturbances, hypophonia, muscle rigidity, postural instability, and dysarthria. This study has investigated the neuroprotective potential of pioglitazone (PIO; a peroxisome proliferator-activated receptor gamma agonist) in attenuating the α-Syn burden and intracellular dysfunction in a rotenone-induced model of PD in rats. To this end, 48 adult male albino Wistar rats were randomly allocated into six groups: a healthy control group, a rotenone-induced PD model group, a vehicle-treated group receiving carboxymethylcellulose as well as rotenone (similarly to the PD model group), a rasagiline-treated group (receiving rasagiline at a dose of 1 mg/kg/day) that also received rotenone (as in the PD model group), and two PIO-treated groups receiving PIO at doses of 10 or 20 mg/kg/day in combination with rotenone (as in the PD model group). Parkinsonism was induced through the intraperitoneal administration of rotenone at a dose of 2.5 mg/kg every 48 h, for 21 days. Oral treatments were initiated 3 days before the first rotenone injection and were continued throughout the experimental period. Body weight was recorded on days 0, 7, 14, and 21. Behavioural assessments were performed on day 22 (24 h after the final treatment dose) using rotarod and open-field tests so as to evaluate locomotor activity, exploratory behavior, and motor coordination. The α-Syn expression was further quantified *via* brain immunohistochemistry and serum ELISA. The rotenone-induced Parkinsonian-like changes were evidenced by an 8.18% reduction in body weight (compared to a 20.35% increase in the control group; $p<0.05$) as well as by a substantial drop in behavioural performance ($p<0.05$) compared to that of the control group. Marked reductions were observed in grooming time (74.17%), number of crossing lines (70.33%), number of rearing episodes (74.84%), and number of visits to the center (85.62%). Motor performance was markedly decreased by rotenone, with decreases observed in the number of rotations (81.23%), the rotation time (69.10%), and the distance of rotation (56.90%) relatively to those of the control group. Addi-

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tionally, increased cerebral α-Syn accumulation (p<0.05) and a 50.09% decrease in the serum α-Syn levels (p<0.05) were recorded in the rotenone-induced PD model group compared to those of the control group. Treatment with PIO exhibited dose-dependent protective effects: high-dose PIO administration significantly improved body weight, resulting in a 12.28% increase of the latter compared to 6.78% and 7.30% increases in the low-dose PIO and the rasagiline groups, respectively. Moreover, the high-dose PIO administration resulted in remarkable increases in the rats' behavioural performance in terms of grooming time (157.06%), crossing lines (192.82%), rearing (183.82%), and center visits (226.54%) as compared to those of the PD model group (p<0.05). Additionally, motor performance was markedly enhanced by the high-dose PIO, as evidenced by an increase in rotation number (199.48%), rotation time (95.12%), and rotation distance (80.59%) relatively to the rotenone-treated PD model group (p<0.05). Finally, the cerebral α-Syn accumulation was found to be reduced by 25.0%, 50.0%, and 75.0% in the low-dose PIO, the high-dose PIO, and the rasagiline groups, respectively (p<0.05), while serum α-Syn levels were found to be increased by 13.11%, 38.27%, and 76.73%, respectively, in these same groups and as compared to the rotenone-induced PD model group's levels (p<0.05). In conclusion, PIO exerted dose-dependent neuroprotective effects on the assessed rotenone-induced Parkinsonism, improving motor performance and reducing central α-Syn burden, with the higher dose producing effects comparable to those of rasagiline.

Keywords

α-Syn; neuroprotection; Parkinson's disease; pioglitazone; rotenone

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COLLOQUIA ERUDITA 2026; 1: 31–32

PROCEEDINGS ABSTRACT

Design, synthesis, characterization, and *in silico* assessment of novel 1,3,4-thiadiazole–urea derivatives as inhibitors of the vascular endothelial growth factor receptor 2

Noora A. Al-Jubouri, Shaker A. Abdul Hussein, and Hussam W. Al-Humadi

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A novel series of 1,3,4-thiadiazole–urea hybrid derivatives was conceived, synthesized, and assessed for their efficacy as vascular endothelial growth factor receptor-2 (VEGFR-2) inhibitors, thereby aiming at a critical mediator of tumour angiogenesis. The synthetic methodology adopted a convergent two-step strategy. Initially, the intermediate, 1-(5-mercapto-1,3,4-thiadiazol-2-yl)-3-(*p*-tolyl)urea (TT), was synthesized through a urea-forming reaction that involved the interaction between 5-amino-1,3,4-thiadiazole-2-thiol and *p*-tolyl isocyanate. Subsequently, the target compounds (i.e., TT-Cl, TT-NO_2, TT-OCH_3, TT-OH, and TT-CH_3) were synthesized through an *S*-alkylation of the free thiol moiety with a variety of substituted phenacyl bromides in an aqueous environment. The chemical structures of these novel 1,3,4-thiadiazole–urea hybrid derivatives were validated through the utilization of Fourier transform infrared (FTIR), proton nuclear magnetic resonance (^{1}H-NMR), and carbon-13 nuclear magnetic resonance (^{13}C-NMR) spectroscopy. The molecular docking investigations were executed by employing the Molecular Operating Environment v.2024.06 software and by targeting the VEGFR-2 kinase domain (PDB ID: 4ASD), with sorafenib serving as the reference standard. The docking technique received validation *via* the successful redocking of the reference ligand, culminating in a root mean square deviation (RMSD) of 1.60 Å. Each of the synthesized derivatives exhibited a promising binding affinity, with docking scores varying from -8.24 to -9.319 kcal/mol, which were determined to be comparable to that of sorafenib (-9.23 kcal/mol). Importantly, the nitro-substituted derivative (TT-NO_2) has risen to the forefront as the most captivating compound, as it exhibited a binding affinity (-9.319 kcal/mol) that narrowly outstripped that of sorafenib. This increased affinity was ascribed to an optimal amalgamation of conserved hydrogen bonding interactions with critical active site residues ASP_{1046} and GLU_{885} in a distance of 2.94 Å and 1.82 Å, respectively; these were further augmented by hydrophobic interactions with LEU_{1019}, ILE_{892}, LEU_{889}, VAL_{916}, VAL_{899}, VAL_{848}, and ALA_{866}. Furthermore, the TT-NO_2 derivative exhibited a significant pi–sulfur interaction with CYS_{1045}, thereby further enhancing its binding stability. Subsequently, and after using the SwissADME web tool for the undertaking of an *in silico* ADME (absorption, distribution, metabolism, and excretion) profiling, it was shown that every compound synthesized followed Lipinski's rule of five with no

Al-Jubouri N. A., Abdul Hussein S. A., Al-Humadi H. W.: Design, synthesis, characterization, and *in silico* assessment of novel 1,3,4-thiadiazole–urea derivatives as inhibitors of the vascular endothelial growth factor receptor 2. *Colloq. Erud.* 1: 31–32 (2026).
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breaches; a fact that strengthens their qualities as potential drugs. The herein studied derivatives also exhibited advantageous physicochemical features, comprising acceptable molecular weights ranging from 398.50 to 429.47 g/mol, hydrogen bond donor numbers ranging from 2 to 3, and acceptor values from 4 to 6, plus a moderate lipophilicity as reflected in a consensus log P ranging from 2.66 to 3.88. Although all compounds demonstrated low gastrointestinal absorption due to their comparatively high topological polar surface area (TPSA; being >137.52 $Å^2$), they also exhibited adequate bioavailability scores (0.55). Notably, none of these derivatives exhibited potential for PAINS (pan-assay interference compounds) alerts or were forecasted to be able to traverse the blood-brain barrier, which could be a beneficial indicator of their ability to not trigger any central nervous system-related adverse effects. However, findings from the profiling of cytochrome P450 inhibition suggest a possible inhibition of CYP2C19, CYP2C9, and CYP3A4 by all studied compounds, thereby emphasizing the necessity for a further investigation into potential drug–drug interactions. Overall, this study presents our findings on a successfully devised and synthesized novel series of thiadiazole–urea derivatives with validated structural integrity; of these derivatives, the nitro-substituted one (TT-NO_2) has emerged as a promising candidate for VEGFR-2 inhibition, exhibiting superior binding affinity through optimal molecular interactions within the kinase active site of the receptor as well as favourable drug-like properties *in silico*.

Keywords

ADME; cancer; molecular docking; 1,3,4-thiadiazole; VEGFR-2

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Conflicts of interest statement

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COLLOQUIA ERUDITA 2026; 1: 33–34

PROCEEDINGS ABSTRACT

Protective effects of a *Capparis spinosa* extract, alone and in combination with silymarin, on the hepatic injury and critical liver function markers of rats exposed to acetaminophen

Abrar Abdulkareem Jawad and Qayssar Joudah Fadheel

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Acetaminophen (APAP) overdose is a major cause of acute drug-induced liver injury worldwide, largely mediated by oxidative stress, lipid peroxidation, and hepatocellular necrosis. Although silymarin is widely used as a hepatoprotective agent, its efficacy may be insufficient in severe intoxication, highlighting the need for effective adjunct therapies. *Capparis spinosa*, a flavonoid-rich medicinal plant with established antioxidant and anti-inflammatory properties, represents a promising candidate for complementary hepatoprotection. In this study, 42 male Wistar rats were randomly assigned to seven groups (n=6 each): a vehicle control group, an APAP-only treatment group (2 g/kg orally, on day 8), an APAP- plus silymarin-treated (100 mg/kg) group, three groups receiving APAP plus a *C. spinosa* ethanolic extract at 100, 200, or 400 mg/kg, and a group receiving APAP as well as a combined treatment with silymarin and the *C. spinosa* extract (200 mg/kg) group. These treatments were administered orally for 10 consecutive days, and animals were sacrificed on day 11. Hepatic injury was assessed through the measurement of the serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, albumin, and total protein levels, in addition to histopathological evaluation using the modified Suzuki score (0–4 scale) for centrilobular necrosis, sinusoidal congestion, and inflammatory infiltration. The undertaken APAP administration induced severe hepatic injury that was characterized by centrilobular necrosis, inflammatory infiltration, and disruption of hepatic architecture, resulting in significantly elevated modified Suzuki scores ($p<0.001$ *versus* control). Serum ALT, AST, ALP, and total bilirubin levels were found elevated by more than 100% above those of the control group, whereas albumin and total protein were found decreased by approximately 52% and 46%, respectively, as a result of the APAP administration ($p<0.001$ *versus* control in all cases). Silymarin treatment markedly attenuated these alterations, by reducing the necrosis score to 0.93 (± 0.10) and by decreasing the serum ALT, AST, ALP, and total bilirubin levels by approximately 71%, 81%, 64%, and 65%, respectively, while increasing those of albumin and total protein by 69% and 46% when compared to those of the APAP-only receiving rats ($p<0.001$ *versus* APAP-only treatment in all cases). The herein employed treatment with the *C. spinosa* ethanolic extract exhibited a dose-dependent hepatoprotective effect. The 100 mg/kg dose reduced the serum

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ALT, AST, ALP, and total bilirubin levels by 48%, 64%, 43%, and 40%, respectively (p<0.001 *versus* APAP-only treatment in all aforementioned cases), whereas the 200 mg/kg dose provided greater biochemical and histological improvement, with reductions of 62%, 77%, 62%, and 58%, respectively in the levels of the aforementioned biochemical parameters (p<0.001 *versus* APAP-only treatment in all cases). Notably, the 400 mg/kg dose of the *C. spinosa* extract restored hepatic architecture and normalized all measured parameters to levels comparable with those achieved by silymarin (p>0.05 *versus* APAP plus silymarin treatment). The combination of silymarin with the *C. spinosa* extract (200 mg/kg) produced the strongest hepatoprotective effect against the APAP-induced hepatotoxicity, yielding the lowest modified Suzuki scores and reducing the serum levels of ALT, AST, ALP, and total bilirubin by 77%, 84%, 69%, and 81%, respectively, relatively to those of the APAP-only treatment group; on the other hand, the albumin and total protein levels were increased by 96% and 64% (p<0.001 *versus* APAP-only treatment in all cases; p<0.05 *versus* APAP plus silymarin treatment in the cases of AST, ALP, and total protein). These findings indicate that the administration of a *C. spinosa* ethanolic extract can exert a significant and dose-dependent hepatoprotection against an APAP-induced liver injury, with the 400 mg/kg dose demonstrating an efficacy that is comparable to that of silymarin.

Keywords

acetaminophen; caper; drug-induced liver injury; hepatotoxicity; silymarin

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COLLOQUIA ERUDITA 2026; 1: 35–36

PROCEEDINGS ABSTRACT

Serum soluble fms-like tyrosine kinase-1 as an ancillary diagnostic biomarker in molar pregnancy: histological correlates and differentiation of a complete from a partial hydatidiform mole in Iraqi women

Mazin J. Mousa and Nareeman Abdulhassan Karbul

Received: 04 April 2026; Revised: 09 June 2026; Accepted: 18 June 2026; Published: 23 June 2026

Hydatidiform moles (HMs) – subclassified into complete (CHMs) and partial (PHMs) ones – represent the commonest gestational trophoblastic disease and carry malignant transformation risks between 15% and 20% and between 0.5% and 5% respectively, making accurate subclassification essential for post-evacuation surveillance. Their histopathological examination remains the diagnostic cornerstone, yet early-evacuation specimens increasingly lack fully developed hallmark features, while ancillary molecular techniques are not universally accessible. Soluble fms-like tyrosine kinase-1 (sFlt-1; also designated sVEGFR-1), a circulating anti-angiogenic splice variant overproduced by syncytiotrophoblasts in molar tissues, represents a candidate serum biomarker, but its quantitative relationship with specific histological parameters of the molar pregnancy and its utility in CHM-*versus*-PHM differentiation had not yet been characterised in a controlled three-group design prior to the present study. This study aimed at evaluating the serum sFlt-1 as an ancillary diagnostic biomarker in HM, at correlating its concentration with binary histological features of molar tissue, and at determining its discriminatory accuracy for CHMs *versus* PHMs and for molar *versus* normal pregnancies in Iraqi women. A cross-sectional observational study was conducted between March 2024 and May 2025 at the Al-Zahrawi Clinical Laboratory (Babylon, Iraq). The patients enrolled in this study were primarily admitted and treated at the Imam Al-Sadiq Teaching Hospital (Babylon, Iraq). A total of 65 subjects were enrolled: 51 women with histologically confirmed HMs (of which 7 with CHMs and 44 with PHMs) and 14 gestational age-matched women with normal pregnancies serving as controls. Uterine evacuation specimens were sent to the Al-Zahrawi Clinical Laboratory for their histopathological examination, and haematoxylin-and-eosin-stained sections were examined independently by two blinded pathologists, who scored seven binary histological parameters. The serum sFlt-1 and beta-human chorionic gonadotrophin (β-hCG) levels were measured in the blood samples (collected at the time of the specimen submission) using the Snibe MAGLUMI 800 fully automated chemiluminescence immunoassay (CLIA) analyser (Snibe Diagnostic, China). Our statistical analysis employed the Kruskal–Wallis and Bonferroni-corrected Mann–Whitney *U* tests, Spearman rank correlations with bootstrap 95% confidence intervals, Fisher's exact test, and receiver operating characteristic analysis with

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Youden's J optimisation. The herein measured serum sFlt-1 levels differed significantly across all three of the studied groups, with median values rising 6.7-fold in PHM cases (1,721.5 pg/mL) and 16.0-fold in CHM cases (4,123.0 pg/mL) over normal controls (257.5 pg/mL; all Bonferroni-corrected at $p<0.001$), with complete separation of distributions. In the case of the molar-*versus*-normal discrimination, the serum sFlt-1 levels achieved an area under the curve (AUC) of 1.000 at a cut-off of ≥1,024 pg/mL (with a sensitivity and a specificity that were both 100%), thereby outperforming the β-hCG levels (AUC of 0.980). In the case of the CHM-*versus*-PHM differentiation, the serum sFlt-1 levels achieved an AUC of 0.997 at a cut-off of ≥3,089 pg/mL (with a sensitivity of 100%, a specificity of 97.7%, and an overall accuracy of 98.0%), with the single false-positive case correctly resolved by the concurrent serum β-hCG levels (AUC of 1.000 at ≥79,120 IU/mL). The CLIA-measured serum sFlt-1 levels correlated significantly ($p<0.001$) with trophoblastic hyperplasia ($r_s=0.54$), cistern formation ($r_s=0.53$), and stromal vascularity ($r_s=-0.55$), thereby mechanistically grounding this biomarker to the histological parameters of trophoblastic overactivity. Our findings establish the automated serum sFlt-1 measurement (through CLIA) as a highly accurate ancillary diagnostic biomarker for molar pregnancy in Iraqi women.

Keywords

gestational trophoblastic disease; hydatidiform mole; β-hCG; sFlt-1; sVEGFR-1

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COLLOQUIA ERUDITA 2026; 1: 37–38

PROCEEDINGS ABSTRACT

Niosomes as versatile nanocarriers: a review of recent application advances and challenges

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This review provides a critical summary of recent advances in the development and application of niosomes as versatile nanocarriers, emphasizing their potential in enhancing drug delivery, improving therapeutic efficacy, and enabling targeted treatment. This review also attempts to highlight the major challenges associated with the formulation and the clinical translation of niosomes, and discusses the future directions for their successful application in nanomedicine. Niosomes are vesicular drug delivery systems formed from non-ionic surfactants that have gained considerable attention as versatile and efficient carriers for modern therapeutics. Structurally, niosomes consist of a bilayer membrane similar to liposomes, but they are more stable and cost-effective due to the use of non-ionic surfactants instead of phospholipids. This bilayer architecture enables the simultaneous encapsulation of both hydrophilic drugs (within the aqueous core) and lipophilic drugs (within the bilayer), making niosomes highly adaptable for a wide range of pharmaceutical applications. One of the key advantages of niosomes over conventional drug delivery systems is their ability to improve the pharmacokinetic and pharmacodynamic profiles of drugs, as they can (i) enhance drug stability by protecting the active compound(s) from degradation, (ii) prolong the drug circulation time in the bloodstream, and (iii) provide controlled or sustained drug release. Additionally, niosomes can increase drug bioavailability, reduce systemic drug toxicity, and even enable targeted or localized drug delivery, which is particularly important for drugs with narrow therapeutic windows or significant side effects. To date, traditional methods of niosome preparation (such as the thin-film hydration and solvent-based techniques) have been widely used due to their simplicity and reproducibility. However, recent advancements have introduced more sophisticated and precise approaches. For example, the implementation of microfluidics allows for a better control over the vesicle size and uniformity, while the adoption of quality-by-design strategies can optimize formulation parameters in order to ensure consistent product quality. Furthermore, the use of surface functionalization has enabled the development of targeted niosomes by attaching to them ligands that recognize specific cells or tissues. Stimuli-responsive systems, which release drugs in response to environmental triggers such as pH, temperature, or enzymes, have further enhanced the therapeutic potential of niosomes. The integration of niosomes into gels or hybrid platforms has also expanded their applicability, particularly in topical and transdermal delivery. These technological advancements have significantly broadened the clinical applications of niosomes, and they are now being explored for oral, topical, transdermal, ocular, and intranasal drug delivery. Furthermore, niosomes exhibit great promise for use in anticancer therapies by improving drug targeting and reducing toxicity to healthy tissues. The niosome use in antimicrobial treatments and vaccine delivery systems

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is also being investigated, as they can enhance immune responses and improve the stability of antigens. Unfortunately, despite these promising developments, several challenges remain in translating niosomal formulations into clinical practice: issues related to large-scale production, batch-to-batch reproducibility, long-term stability, sterilization, and regulatory standardization still need to be addressed. Of these, ensuring consistent quality and efficacy of niosomes across different manufacturing scales is particularly critical for commercial viability. In conclusion, niosomes represent a highly versatile and rapidly evolving platform for drug delivery. Given the ongoing innovations in formulation design and manufacturing technologies, they hold significant potential to bridge the gap between experimental research and clinically effective therapies, ultimately being able to improve patient outcomes across a wide range of medical conditions.

Keywords

drug delivery; nanocarriers; niosomes; non-ionic surfactant; targeted therapy

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COLLOQUIA ERUDITA 2026; 1: 39–40

PROCEEDINGS ABSTRACT

The translational research potential of Iraq's native fauna: the desert gerbil (*Psammomys obesus*) as a non-genetic model of diet-induced metabolic syndrome progression

Noor Dheyaa Kadhim, Apostolos Zarros, Hussam W. Al-Humadi, and Rafal J. Al-Saigh

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The growing global burden of the metabolic syndrome and of its complications necessitates the development of high-fidelity animal models that can accurately replicate the associated human pathophysiology. To this end, transgenic models frequently fail to capture the complex environment–gene interactions characteristic of the human disease. We herein review recent findings and highlight the translational research importance of the fat sand rat *Psammomys obesus* (a desert gerbil species that is indigenous to the arid environments of Iraq) as a non-genetic model of diet-induced metabolic syndrome progression. *Psammomys obesus* maintains a lean phenotype when in its natural habitat, as it has adapted to survive on a nutrient-poor, low-calorie halophytic diet; however, when this species is transitioned to a standard, high-calorie laboratory diet, it exhibits a cascade of metabolic derangements. This response is believed to mirror that of human populations undergoing nutritional transition as part of the adoption of a (more) Westernized lifestyle, thereby offering a diet-induced, polygenic platform for studying human diseases such as type 2 diabetes mellitus, non-alcoholic fatty liver disease (NAFLD), cardiovascular remodelling, and the metabolic syndrome at large. Our review synthesizes established and emerging evidence detailing the multi-organ effects of dietary interventions in *Psammomys obesus* [1]. The metabolic syndrome-simulating pathophysiological trajectory in this species follows a well-characterized progression from an early compensatory hyperinsulinaemia to a terminal pancreatic β-cell exhaustion and an absolute insulin deficiency [2]. Notably, *Psammomys obesus* can develop "clinical" complications that are notoriously difficult to reproduce in genetically modified rodent models. For example, within the cardiovascular system, the diet-induced stress has been shown to precipitate arterial wall thickening, profound myocardial fibrosis, and severe endothelial dysfunction. Concurrently, the liver of this desert gerbil species is capable of undergoing a rapid diet-induced progression toward advanced NAFLD, that is characterized by aggressive steatosis and irreversible fibrosis. Studies have also highlighted the model's importance for the simulation and elucidation of metabolic syndrome-associated microvascular pathologies, including spontaneous diabetic retinopathy (marked by retinal microvascular permeability) and diabetic nephropathy (featuring significant glomerular basement membrane thickening and overt albuminuria). This unique suscep-

Kadhim N. D., Zarros A., Al-Humadi H. W., Al-Saigh R. J.: The translational research potential of Iraq's native fauna: the desert gerbil (*Psammomys obesus*) as a non-genetic model of diet-induced metabolic syndrome progression. *Colloq. Erud.* 1: 39–40 (2026).
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tibility of *Psammomys obesus* to diet-induced metabolic collapse is believed to be driven by uncontrolled oxidative stress and the aberrant expression of key glucose transporters. In fact, unlike traditional laboratory rodents, this species possesses an inherently limited endogenous antioxidant capacity; therefore, when *Psammomys obesus* is exposed to a high-calorie diet, this inherent deficit leads to an accumulation of reactive oxygen species as well as to an accelerated mitochondrial dysfunction across many of the species' tissues. By capturing these diverse systemic manifestations alongside this molecular vulnerability, the diet-induced *Psammomys obesus* model provides a comprehensive representation of the metabolic syndrome's progression that closely simulates clinical reality and offers an invaluable experimental platform for the validation of cytoprotective and metabolism-modulating agents [3]. From a regional perspective, the harnessing of the translational research potential of *Psammomys obesus* may hold strategic importance for the Iraqi scientific community. As a species that is native to Iraq, it represents an accessible and cost-effective biological resource that circumvents many of the logistical and financial burdens associated with importing and maintaining specialized transgenic models. The systematic use of this species in Iraq as a non-genetic model of diet-induced metabolic syndrome progression could facilitate, amongst other translational research applications, the pharmacological screening of bioactive compounds deriving from native flora, thereby strengthening the country's position as a drug discovery hub in the Middle East.

Keywords

desert gerbil; diabetes; Iraq; metabolic syndrome; translational research

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Conflicts of interest statement

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PROCEEDINGS ABSTRACT

Antifungal efficacy of selected oral antidiabetic drugs against *Candida albicans* and *Candida glabrata*: an *in vitro* pharmacodynamic analysis

Duha Hussain Abd-Alhadi and Hussam W. Al-Humadi

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Type 2 diabetes mellitus (T2DM) is a chronic, progressive metabolic disorder that can trigger profound systemic immune dysfunction, thereby markedly increasing patient susceptibility to severe opportunistic infections. Chronic hyperglycaemia can impair critical host defence mechanisms; as a result, diabetics face a substantially elevated risk of both mucosal and invasive fungal infections, most notably infections caused by a *Candida* species. Although *Candida albicans* remains the predominant isolated pathogen, other species such as *Candida glabrata* are rapidly emerging due to their innate resilience in glucose-rich environments and their baseline resistance to standard antifungal therapies [1]. Concurrently, increasing amount of evidence suggests that common oral antidiabetic drugs (OADs) that are traditionally utilized solely for glycaemic control, may also possess secondary, off-target, antimicrobial properties. This study aimed at quantifying and comparing the intrinsic *in vitro* antifungal efficacies of five widely prescribed OADs (metformin, pioglitazone, gliclazide, vildagliptin, and dapagliflozin) against standard clinical isolates of *Candida albicans* (ATCC-90028) and *Candida glabrata* (ATCC-90300). The drugs' antifungal activity was initially assessed by employing a highly controlled 96-well static microdilution assay. Serial concentrations of each OAD (0.125–8.0 mg/L) were inoculated with a standardized fungal suspension (2×10^4 CFU/mL) in an RPMI-1640 growth medium. After a 24-h incubation at 37°C, the relative optical density was quantified spectrophotometrically at 600 nm in order to measure dynamic fungal growth inhibition. In an attempt to overcome the inherent limitations of static concentration models and to accurately simulate the *in vivo* physiological drug clearance, a dynamic *in vitro* pharmacokinetics / pharmacodynamics (PK/PD) model was employed using a precision peristaltic pump and a cellulose dialysis membrane (<50-kDa). The system replicated continuous human plasma clearance and measured the cumulative drug exposure–response relationship. Comprehensive PD parameters, specifically the half-maximal inhibitory concentration (IC_{50}), maximum inhibitory effect (E_{max}), and area under the curve (AUC), were calculated using a nonlinear variable-slope sigmoidal regression model. The mathematical modelling demonstrated exceptional robustness, with goodness-of-fit (R^2) values exceeding 0.973 across all evaluated dose–response curves. Metformin exhibited the most potent intrinsic antifungal profile, achieving an E_{max} of 72% (IC_{50}: 1.0 mg/L; AUC: 82.85%) against *C. albicans* as well as of 68% (IC_{50}: 2.0 mg/L; AUC: 76.40%) against *C. glabrata*. Pioglitazone exhibited robust secondary efficacy, yielding an E_{max} of 65% (IC_{50}: 2.0 mg/L) against *C. albicans* and of 56% (IC_{50}: 4.0 mg/L) against *C. glabrata*. Gliclazide showed moderate but consistent fungistatic

Abd-Alhadi D. H., Al-Humadi H. W.: Antifungal efficacy of selected oral antidiabetic drugs against *Candida albicans* and *Candida glabrata*: an *in vitro* pharmacodynamic analysis. *Colloq. Erud.* 1: 41–42 (2026).
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properties across the two strains (achieving an E_{max} of 50% and 45%, respectively). On the other hand, vildagliptin and dapagliflozin exhibited the weakest antifungal activities, both requiring the maximum tested concentration (IC_{50}: 8.0 mg/L) in order to achieve minimal inhibition thresholds (peak E_{max} of 42% and 40%, respectively, against *C. albicans*). The employed dynamic PK/PD model has confirmed that a sustained drug exposure is directly correlated with compounding antifungal responses, particularly in the case of metformin. Our study provides quantitative confirmation that specific OADs possess highly variable but biologically significant, intrinsic antifungal properties. In fact, metformin and pioglitazone demonstrate substantial fungistatic efficacy at concentrations approaching clinically relevant plasma levels. Conversely, the remarkably poor intrinsic antifungal activity of dapagliflozin supports the current clinical consensus that the sodium-glucose co-transporter-2 (SGLT2) inhibition-associated candidiasis is primarily an environmental consequence of induced glycosuria that creates a nutrient-rich environment for fungal growth rather than a direct immune or structural failure induced by the SGLT2 inhibitors themselves [2,3]. The antifungal effects of these specific agents may offer a secondary clinical advantage against opportunistic *Candida* infections in vulnerable T2DM populations. Further *in vivo* studies are critically warranted in order to translate these dynamic PD findings into proactive clinical practice.

Keywords

antifungal; *in vitro*; metformin; oral antidiabetic drugs; pharmacodynamics

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COLLOQUIA ERUDITA 2026; 1: 43–44

PROCEEDINGS ABSTRACT

Efficacy of non-steroidal anti-inflammatory drugs in the pain management of acute renal colic and irritable bowel syndrome: evidence from an Iraqi emergency department

Ali Jalil Al-Saigh, Hala Saad Bash, Suhad Hussein Atshan, Rafal J. Al-Saigh, and Sally H. Al-Humadi

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Pain is a primary driver of emergency department (ED) visits. Two high-burden but pathophysiologically distinct causes of pain are acute renal colic (ARC) and the irritable bowel syndrome (IBS). Of these, ARC presents with acute, prostaglandin-mediated inflammatory pain as a result of ureteral obstruction [1], whereas IBS features functional pain driven by visceral hypersensitivity and gut-brain dysregulation [2]. Despite these differences, both require a rapid, effective, opioid-sparing analgesia in the ED. The intravenous non-steroidal anti-inflammatory drugs (NSAIDs), such as ketorolac, are standard first-line therapies that effectively target the prostaglandin synthesis that is central to the pathophysiology of the ARC-induced pain. However, their efficacy in treating the non-inflammatory pain of IBS remains poorly defined. Currently, there is a significant lack of comparative clinical data evaluating the NSAID performance across these two distinct clinical entities; addressing this knowledge gap is essential, particularly in resource-constrained healthcare systems where most treatment decisions heavily depend on drug availability, cost, and speed of onset. This prospective study has aimed at evaluating and comparing the clinical efficacy of intravenous NSAIDs in managing acute abdominal and flank pain among patients presenting with ARC *versus* IBS. The investigation was conducted within an Iraqi ED setting at the Imam Al-Hujjah Hospital (Karbala, Iraq) over a 12-month period (from June 2024 to May 2025). The primary objective was to quantify the exact temporal dynamics of the analgesic response. A total of 178 adult patients was prospectively enrolled and stratified into two groups based on confirmed clinical diagnosis. The first comprised 91 patients presenting with ARC (52 males and 39 females, with a mean age of 42.4 years), while the second cohort included 87 patients with an established diagnosis of IBS experiencing acute exacerbations (35 males and 52 females, with a mean age of 39.1 years). Pain intensity was objectively assessed using a standardized 100-mm paper-based visual analogue scale (VAS). Baseline pain scores were recorded upon the ED arrival, followed by serial evaluations at continuous 15-min intervals immediately following pharmacological intervention. All participants received a uniform protocol of intravenous ketorolac as the definitive first-line analgesic agent, and the primary outcome measured was the median time required to achieve clinically significant pain relief, defined *a priori* as a minimum 50% reduction in the initial VAS score.

Al-Saigh A. J., Bash H. S., Atshan S. H., Al-Saigh R. J., Al-Humadi S. H.: Efficacy of non-steroidal anti-inflammatory drugs in the pain management of acute renal colic and irritable bowel syndrome: evidence from an Iraqi emergency department. *Colloq. Erud.* 1: 43–44 (2026).
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Secondary outcomes included the absolute magnitude of pain score reduction at 1 h and the cumulative proportion of patients successfully achieving predefined pain relief thresholds. Results demonstrated that intravenous ketorolac provided rapid and clinically meaningful analgesia across both diagnostic cohorts; however, statistically significant variations were also observed regarding the onset speed and trajectory of pain relief. Patients with IBS exhibited a significantly faster analgesic response [3], with 82.8% achieving substantial pain reduction within the 15- to 30-min observation window (p<0.001). Furthermore, the mean VAS reduction for the IBS group at 30 min was notably higher compared to that of the ARC group. In distinct contrast, the analgesic effect in the ARC cohort was significantly delayed, as 90.2% of these patients required between 30 and 45 min to reach comparable levels of clinically significant relief (p<0.001). The absolute reduction in baseline VAS scores measured at the 60-min mark was substantial in both groups, yet marginally superior in the ARC group. In conclusion, intravenous ketorolac serves as a highly effective, opioid-sparing analgesic for managing acute pain presentations in the ED across both the ARC and IBS populations, though the distinct temporal response dynamics necessitate tailored clinical expectations so as to optimize outcomes.

Keywords

acute renal colic; emergency department; irritable bowel syndrome; NSAIDs; pain management

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Conflicts of interest statement

None to declare.

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COLLOQUIA ERUDITA 2026; 1: 45–46

PROCEEDINGS ABSTRACT

C3 and C4 complement consumption patterns as predictors of haemolysis severity in Iraqi autoimmune haemolytic anaemia patients: a study employing a multimarker integration approach

Zaman I. L. Al-Kaabi

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Autoimmune haemolytic anaemia (AIHA) is a clinically heterogeneous disorder characterized by an immune system-mediated destruction of erythrocytes, primarily driven by autoantibodies and frequently amplified by complement activation. Although conventional laboratory parameters such as haemoglobin, lactate dehydrogenase (LDH), bilirubin, and haptoglobin levels remain essential for assessing an haemolysis, they do not adequately capture the underlying immunopathological mechanisms that influence disease severity. Increasing attention has therefore been directed toward the complement system (particularly its classical pathway) as a key mediator of erythrocyte destruction and a potential source of clinically relevant biomarkers. In this context, the present study (that was conducted between October 2024 and September 2025 at the Haematology Department of the Merjan Teaching Hospital, Hillah, Iraq) aimed at evaluating the complement consumption patterns (specifically those of complement C3 and C4) and at determining their predictive value for haemolytic severity in AIHA by using a multimarker integration approach. A total of 40 Iraqi patients with AIHA were included in this study (55% females; mean age of 42.6 ± 14.3 years); these patients were categorized under mild (30%), moderate (50%), or severe (20%) haemolysis according to their haemoglobin levels. Our findings exhibit a clear severity-associated pattern across the herein assessed haematological and biochemical parameters. More specifically, the haemoglobin levels declined progressively from 10.8 ± 0.9 g/dL in mild cases to 6.9 ± 0.7 g/dL in severe cases (p<0.001), while the reticulocyte counts increased from 3.2% ± 0.9% to 11.4% ± 2.1%, respectively. Similarly, the serum LDH levels rose from 320 ± 85 U/L to 910 ± 180 U/L, while the indirect bilirubin ones were found increased from 1.2 ± 0.4 mg/dL to 4.6 ± 1.1 mg/dL (p<0.001). The herein undertaken complement profiling revealed reduced C4 levels in 65% of the patients and reduced C3 levels in 45% of them, with a combined depletion being observed in 37.5% of the cohort and in up to 75%–80% of the severe AIHA cases. Correlation analysis exhibited significant inverse relationships between complement levels and haemolytic markers (C3 *vs.* LDH levels: r=-0.62; C4 *vs.* LDH levels: r=-0.68; p<0.01), alongside positive correlations with the haemoglobin levels (C3 levels: r=0.58; C4 levels: r=0.63). The identified strong positive correlation between C3 and C4 (r=0.72, p<0.001) – which was found increased to 0.81 in severe haemolysis cases – suggests a coordi-

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nated activation of the classical complement pathway. Moreover, multivariate logistic regression has identified the reticulocyte count as an independent predictor of severe haemolysis in AIHA (OR=1.88, 95% CI: 1.18–2.97, p=0.006), whereas the complement components displayed strong, yet non-independent associations. In the undertaken receiver operating characteristic analysis, a moderate diagnostic performance for individual markers (C3: AUC=0.74; C4: AUC=0.78) was identified, with the C4 levels showing higher sensitivity (82%) and the LDH ones showing higher specificity (85%). Notably, the combined multimarker model integrating C3, C4, and LDH levels is characterized by improved diagnostic accuracy (AUC=0.88), achieving balanced sensitivity (85%) and specificity (83%). Finally, the assessed multimarker scoring system has demonstrated a stepwise increase in the probability of severe haemolysis in AIHA, rising from less than 10% in the low-score categories to more than 75% in the high-score ones. In conclusion, the complement consumption patterns, particularly the combined depletion of C3 and C4, are closely associated with haemolytic severity in Iraqi AIHA patients; to this end, the integration of such parameters with conventional laboratory markers can significantly enhance diagnostic performance, provide a practical framework for risk stratification, and even guide clinical decision-making and therapy.

Keywords

autoimmune haemolytic anaemia; C3 complement; C4 complement; complement consumption; Iraq

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PROCEEDINGS ABSTRACT

Ocular drug delivery: a review of emerging approaches and advances

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Ocular drug delivery still presents a major pharmaceutical challenge. Structures such as the corneal epithelium, the conjunctiva, the tear film, and the nasolacrimal drainage system, all restrict drug diffusion and shorten drug retention times, while quick blinking and high-speed tear turnover can dilute the applied drugs and remove them, resulting in poor drug absorption. Furthermore, the static and dynamic barriers such as the blood–aqueous and the blood–retinal barriers restrict drug penetration into deeper ocular tissues, particularly the posterior segment. These factors collectively contribute to a low therapeutic efficiency and necessitate frequent dosing. The aim of this review is to comprehensively assess conventional ophthalmic formulations alongside advanced, emerging drug delivery systems, so as to highlight recent progress in overcoming the anatomical barriers of the eye, enhancing therapeutic efficacy, and improving patient compliance. Conventional ophthalmic dosage forms, including eye drops, suspensions, and ointments, are widely used in clinical practice due to their ease of administration and patient acceptance. However, these formulations are associated with significant disadvantages. For example, the bioavailability of topically applied agents is typically very poor, often less than or equal to 5%, due to rapid precorneal loss and the lack of corneal permeability. Suspensions, which may enhance drug retention time, often induce irritation by making the cornea shift or by giving rise to inconsistent dosing patterns. Ointments, whilst providing a prolonged contact with the ocular surface, impair vision and hinder patient acceptability. These conventional systems lack the ability to facilitate the drug penetration into the posterior segment of the eye, thereby precluding their use in diseases such as the age-related macular degeneration and diabetic retinopathy. Consequently, invasive treatments in the form of intravitreal injections are often necessary, introducing the likelihood of causing an infection, retinal damage, and discomfort to the patient. In order to overcome these limitations, novel advanced drug delivery systems have been developed with the aim of enhancing ocular bioavailability, prolonging drug residence time, and enabling targeted delivery. Nanotechnology-based carriers (namely, nanoparticles, liposomes, and nanoemulsions) offer improved permeability and controlled drug release, thereby allowing the carried drugs to penetrate ocular barriers more effectively. The surface modification of nanoparticles can further enhance mucoadhesion and cellular uptake, consequently improving therapeutic outcomes. Another promising approach involves the use of microneedle-based systems, enabling a minimally invasive delivery of drugs to both the anterior and the posterior segments while reducing the risks associated with conventional injections. *In situ* gels, implants, and biodegradable depots can also provide sustained drug release, thereby minimizing dosing frequency and improving patient compliance. Furthermore, emerging drug delivery strategies are now

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exploring systemic drug delivery to the eye by modulating physiological barriers such as the blood–retinal barrier and enabling drugs administered *via* systemic routes to achieve therapeutic concentrations at the ocular tissues. Such an advanced ocular drug delivery system can be realized through the use of ligand-receptor interactions and cell-penetrating peptides that enhance drug uptake by specific ocular tissues. Novel drug delivery strategies employing methods involving the use of antibody–drug conjugates, aptamer-based targeting, and receptor-mediated endocytosis can help drugs accumulate and target disease-affected cells while sparing healthy ones; this selectivity is particularly valuable when treating retinal diseases and intraocular tumours. Despite promising progress, challenges related to safety, scalability, and regulatory approval remain, necessitating continued interdisciplinary research in order to translate these innovations into clinical practice. In this context, artificial intelligence could prove very helpful in designing treatments and predicting responses.

Keywords

advanced drug delivery systems; bioavailability; eye permeability; nanoparticles; ocular drug delivery

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COLLOQUIA ERUDITA 2026; 1: 49–50

PROCEEDINGS ABSTRACT

Protective effects of herbal extracts against drug-induced toxicity: a review of pharmacological evidence and mechanisms

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Drug-induced toxicity is a major challenge in pharmacotherapy and is considered one of the leading causes of treatment interruption, hospitalization, and progressive organ dysfunction worldwide. Several commonly prescribed drugs (including acetaminophen, cisplatin, methotrexate, and doxorubicin) are well-known to exert toxic effects on organs such as the liver, the kidneys, and the heart, and although such drugs might differ in their pharmacological actions and therapeutic applications, they can frequently induce tissue injury through overlapping molecular mechanisms. In fact, oxidative stress, mitochondrial dysfunction, the activation of inflammatory signalling, and apoptosis are considered amongst the most important pathogenic pathways involved in drug-induced toxicity. Excessive generation of reactive oxygen species disrupts intracellular redox balance and promotes lipid peroxidation, protein oxidation, DNA damage, and impairment of normal cellular metabolism. Additionally, mitochondrial injury contributes to ATP depletion and the activation of apoptotic pathways, while the inflammatory mediators further amplify tissue injury and accelerate organ dysfunction. The acetaminophen-induced hepatotoxicity is primarily linked to the formation of a highly reactive metabolite that depletes intracellular glutathione stores and induces severe oxidative stress in the hepatocytes. Similarly, the cisplatin-induced nephrotoxicity involves an excessive production of reactive oxygen species, inflammatory cytokine release, mitochondrial injury, and structural damage to the renal tubular cells. The doxorubicin-induced cardiotoxicity has been linked to mitochondrial dysfunction, iron-mediated oxidative reactions, calcium imbalance, and apoptosis of cardiomyocytes, ultimately leading to impaired cardiac performance and irreversible cardiac injury in severe cases. These overlapping pathogenic pathways emphasize the central role of oxidative and inflammatory mechanisms in the progression of drug-induced toxicity across multiple tissues and organ systems. In recent years, herbal extracts and phytochemicals have attracted considerable interest as potential protective agents against drug-induced toxicity. Numerous bioactive compounds isolated from medicinal plants have demonstrated antioxidant, anti-inflammatory, antiapoptotic, and cytoprotective properties in experimental studies. For example, silymarin has been reported to stabilize cellular membranes, enhance antioxidant enzyme activity, and reduce lipid peroxidation in hepatocytes, while curcumin has been shown to exert potent anti-inflammatory effects through the inhibition of the nuclear factor kappa-B signalling pathways and the suppression of inflammatory mediator production. Another example is epigallocatechin gallate (the major catechin found in green tea), which has been shown to act as a powerful free

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radical scavenger and to be able to protect macromolecules from oxidative injury. Finally, thymoquinone has been shown to exhibit both antioxidant as well as anti-inflammatory properties that can counteract the tissue damage caused by toxic metabolites, whereas ginsenosides have been shown to improve mitochondrial function, regulate apoptosis-related pathways, and enhance endogenous antioxidant defences. Experimental studies suggest that these phytochemicals may attenuate tissue injury, preserve mitochondrial integrity, improve biochemical markers of organ function, and enhance cellular resistance against toxic insults. Interestingly, several studies also indicate that herbal compounds may support cellular recovery processes and improve tissue regeneration following drug-induced toxicity. This review summarizes the principal molecular mechanisms underlying drug-induced toxicity and critically evaluates current evidence regarding the protective effects of selected herbal extracts and phytochemicals, while discussing their potential therapeutic significance and the existing limitations for their clinical application.

Keywords

drug toxicity; herbal extracts; mitochondrial dysfunction; oxidative stress; phytochemicals

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PROCEEDINGS ABSTRACT

In vitro pharmacodynamic modelling of the antibacterial effects of selected oral antidiabetic drugs against *Staphylococcus aureus* and *Escherichia coli*

Duha Hussain Abd-Alhadi and Hussam W. Al-Humadi

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Type 2 diabetes mellitus (T2DM) represents a profound global health burden that is associated with a heightened susceptibility to localized and systemic bacterial infections. This enhanced risk profile is primarily driven by chronic hyperglycaemia-induced immune dysfunctions (including compromised neutrophil chemotaxis and impaired phagocytic activity), and managing infectious morbidities in diabetic patient cohorts remains a clinical challenge. Emerging evidence suggests that certain classes of oral antidiabetic drugs (OADs) possess pleiotropic, intrinsic antimicrobial properties extending beyond their primary role in glycaemic regulation. This study has aimed at rigorously assessing and comparatively quantifying the *in vitro* antibacterial efficacy of five structurally distinct OADs (namely, metformin, pioglitazone, gliclazide, vildagliptin, and dapagliflozin) against two major opportunistic human pathogens: *Staphylococcus aureus* and *Escherichia coli*. In an attempt to fully characterize these antibacterial profiles, a standardized *in vitro* methodological approach was employed. Antibacterial activity was quantitatively assessed utilizing a high throughput 96-well microdilution assay. Pathogen isolates were exposed to a calibrated, clinically relevant concentration gradient of the selected OADs, ranging from 0.125 to 8.0 mg/L, while bacterial proliferation was dynamically quantified by measuring the relative optical density at regular intervals. In order to translate empirical observations into robust quantitative metrics, the herein obtained concentration–effect datasets were subjected to advanced pharmacodynamic (PD) modelling. In fact, a sigmoidal maximum achievable inhibitory effect (E_{max}) model was applied so as to accurately estimate critical PD parameters, encompassing the half maximal inhibitory concentration (IC_{50}), E_{max}, as well as the comprehensive area under the effect curve (AUC). The PD modelling yielded highly robust fits that were validated by stringent goodness-of-fit coefficients (R^2>0.96), thereby ensuring the reliability of the derived parameters. The comparative analysis revealed striking divergences in antimicrobial potency amongst the tested OADs. In fact, metformin unequivocally demonstrated the highest antibacterial efficacy across the pathogen spectrum, achieving an impressive E_{max} of 70% against *S. aureus* and peaking at 78% against *E. coli*, while simultaneously generating the largest AUCs [1]. Gliclazide and vildagliptin exhibited moderate, yet notable, bacteriostatic activity. On the other hand, pioglitazone displayed the most attenuated effect against the Gram-positive *S. aureus* (with a minimal E_{max} of 28%), yet interestingly maintained a moderate inhibitory activity against the Gram-negative *E. coli* (with an E_{max} of 62%). A critical overarching trend in our experimental findings was the distinctly greater susceptibility of the Gram-negative

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strain: collectively, lower IC_{50} thresholds (predominantly converging at OAD concentrations of 2.0 mg/L) were required in order to actively inhibit *E. coli* compared to *S. aureus*, thereby suggesting specific target vulnerabilities related to different cell envelope architectures. Our *in vitro* study suggests that widely prescribed OADs harbour variable but highly quantifiable intrinsic antibacterial properties against foundational human pathogens [2,3]. The superior *in vitro* potency of metformin, followed by that of gliclazide and of vildagliptin, suggests a compelling secondary pharmacological benefit. While these molecules remain categorically defined by their vital role in metabolic management, their latent antimicrobial capacity could offer a clinical advantage in mitigating the severity of bacterial infections in vulnerable T2DM patient populations.

Keywords

antibacterial activity; gliclazide; *in vitro* pharmacodynamics; metformin; oral antidiabetic drugs

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COLLOQUIA ERUDITA 2026; 1: 53–54

PROCEEDINGS ABSTRACT

Impact of the COVID-19 vaccination on hair loss: a study on vaccinated individuals from the Babil Province (Iraq)

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The coronavirus disease 2019 (COVID-19) mainly leads to symptoms affecting the lower respiratory tract and causes pulmonary complications such as the acute respiratory distress syndrome; it is now established as a multisystem disease exhibiting diverse manifestations. This retrospective survey aimed at determining whether there is an impact of the COVID-19 vaccination on hair loss in vaccinated Iraqi individuals. The study was conducted online, and the questionnaire was distributed through electronic media, with all data being collected exclusively through the Google Forms platform. The study was carried out from October to March 2024 and was limited to Iraq's Babylon Governorate (including its districts and sub-districts). The study included a total of 221 individuals who had received at least one dose of a COVID-19 vaccine in the past; of these participants, 78 were men and 143 were women. The average age of the participants was 26.61 ± 6.89 years, with an age range of 20 to 70 years. Our findings indicated that a significant majority of respondents (62.5%) were from the city center (Hillah), and it was also noted that 95% of the respondents did not have any comorbidity. Our data revealed that the majority of respondents (92.3%) were within the 20- to 30-year age bracket, with a higher proportion of the participants being women (64.7%). Post vaccination hair loss was found to be significantly higher among females when compared to males (34.1% *versus* 65.9%, $p<0.05$). Our data also suggest that approximately three quarters (73.7%) of the study's participants had been vaccinated with the Pfizer–BioNTech COVID-19 vaccine, followed by those vaccinated with the Sinopharm BIBP COVID-19 vaccine (10.4%) or the Oxford–AstraZeneca one (8.6%). A significant correlation was identified between receiving a COVID-19 vaccine and hair loss in both sexes. In fact, hair loss was quite common among our study's participants, with 47 (22.9%) out of 221 of those who had received a COVID-19 vaccine reporting hair loss following the first or the second dose. The lowest hair loss rates amongst men and women based on comorbidity status were 5.1% and 7.7%, respectively. This was consistent with the very low number of participants in our study reporting comorbidities compared to healthy participants, while no significant differences were noted between men and women regarding such comorbidities (namely, diabetes mellitus, hypertension, and allergies). Interestingly, no significant differences ($p>0.05$) were recorded between the different types of vaccines with regard to hair loss-triggering in Iraqi men or women. Our study has also identified the most common types of hair loss, with a higher prevalence in females than in males for alopecia areata (70.9%) and androgenetic alopecia (62.5%). In conclusion, hair loss may be associated with COVID-19 vaccination, is more frequent in women than men, and

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may be influenced by comorbidities such as diabetes mellitus and allergies.

Keywords

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PROCEEDINGS ABSTRACT

Anti-inflammatory and antioxidant effects of medicinal plant extracts: a review of pharmacological evidence and mechanisms

Safa Adnan Mahmood, Qayssar Joudah Fadheel, and Karar M. Jaleel

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Several non-communicable diseases (including cardiovascular disorders, type 2 diabetes mellitus, most neurodegenerative diseases, autoimmune conditions, and cancer) are now known to be directly related to chronic inflammation and oxidative stress. Although such diseases significantly vary in their clinical manifestations, they tend to share common pathophysiological pathways that involve an excessive production of reactive oxygen species (ROS) and a persistent activation of the inflammatory signalling. The excessive ROS generation damages lipids, proteins, and DNA, and activates pro-inflammatory transcription factors such as the nuclear factor-kappa B (NF-κB) and the activator protein-1. The activation of these molecules increases the expression of inflammatory mediators such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6). This creates a self-perpetuating cycle in which oxidative stress promotes inflammation and the latter generates additional ROS, leading to tissue injury and disease progression. In this broad context, medicinal plant extracts have gained considerable attention due to their ability to simultaneously influence multiple biological targets. Plant extracts are known to be rich in bioactive phytochemicals (such as polyphenols, flavonoids, terpenoids, alkaloids, and phenolic acids), and these compounds seem to possess antioxidant and anti-inflammatory properties through several complementary mechanisms. For example, a significant number of phytochemicals can directly counteract free radicals and ROS: they may have the ability to chelate transition metals such as iron and copper, which otherwise catalyse the formation of more free radicals. They can also prevent lipid peroxidation, thereby protecting the cellular membranes from oxidative damage. In addition to these direct effects, plant-derived compounds can strength the body's own defence systems by increasing the activities of endogenous antioxidant enzymes (such as superoxide dismutase, catalase, and glutathione peroxidase). This effect is mainly mediated through the activation of the nuclear factor erythroid 2-related factor 2 / antioxidant response element signalling pathway, which controls the expression of genes involved in antioxidant protection. Additionally, medicinal plant extracts can inhibit key inflammatory pathways: numerous phytochemicals are known to be able to suppress the expression of cyclooxygenase-2 and of the inducible nitric oxide synthase; two enzymes that are strongly associated with inflammatory damage. Some phytochemicals can also prevent the nuclear translocation of NF-κB, thereby lowering the generation of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. Data obtained from cell culture and animal model studies strongly support these positive effects. Furthermore, a growing number of randomized

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controlled trials involving the use of medicinal plants (such as *Curcuma longa*, *Camellia sinensis*, and *Zingiber officinale*) has shown reductions in biomarkers of oxidative stress and inflammation in humans. Despite these promising findings, several limitations hamper clinical application: numerous phytochemicals have poor bioavailability (meaning that only a small amount reaches the bloodstream after administration), while plant extracts often lack standardisation, resulting in variable compositions and potencies. Moreover, large scale clinical trials and long-term safety studies remain limited. Further mechanistic research as well as appropriately designed evidence-based clinical trials are needed before medicinal plant extracts can be systematically integrated into modern pharmacotherapy.

Keywords

chronic inflammation; medicinal plant extracts; NF-κB; oxidative stress; phytochemicals

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PROCEEDINGS ABSTRACT

Pathophysiological mechanisms and emerging pharmacological strategies in sepsis-associated multiple organ dysfunction: a review

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Sepsis is a life-threatening syndrome that arises when the host response to an infection becomes dysregulated and injurious rather than protective. Instead of containing the infection, the immune, vascular, and metabolic responses begin to damage host tissues, leading to progressive dysfunction in multiple organs. This process remains a major cause of death in critically ill patients despite advances in intensive care practice, antimicrobial therapy, and organ support. The burden of sepsis is particularly high in patients with a delayed diagnosis, severe comorbidities, advanced age, or a persistent shock, and mortality rises markedly once failure develops in more than one organ system. Its clinical course is often heterogeneous, which partly explains why many promising interventions have not translated successfully from experimental models into routine clinical care across diverse patient populations. The biological basis of the sepsis-associated multiple organ dysfunction is highly complex. It involves an early surge of inflammatory mediators triggered by both pathogen- and damage-associated molecular patterns, that is followed by immune exhaustion or immunosuppression. Endothelial injury, microcirculatory failure, disturbed coagulation, mitochondrial dysfunction, and an altered cellular metabolism contribute to the impaired tissue oxygen use and the progressive cellular damage. These changes affect the liver, the lungs, the heart, the kidneys, and other organs through overlapping mechanisms rather than through isolated pathways. Hepatic dysfunction may reflect cytokine-driven injury, cholestasis, and impaired perfusion; cardiac dysfunction is linked to inflammatory signalling, mitochondrial injury, and reduced contractile performance; acute lung injury develops through an alveolar–capillary barrier disruption and neutrophil recruitment; acute kidney injury involves inflammation, microvascular disturbance, oxidative stress, and tubular dysfunction. Due to the fact that the aforementioned organ-specific manifestations share common pathophysiological networks, sepsis should be viewed as a systemic disorder rather than a series of distinct complications. Current management still largely depends on the rapid recognition, the timely (broad-spectrum) antimicrobial therapy, source control, haemodynamic stabilization, and intense organ-supportive care. Although these measures are essential, they do not directly reverse any of the molecular disturbances that drive organ injury. For this reason, increasing attention has turned toward mechanism-based therapies that can interrupt disease progression. In fact, recent preclinical studies have explored several pharmacological agents that exhibit antioxidant, anti-inflammatory, immunomodulatory, and even mitochondria-protective

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properties, including melatonin, metformin, and palmitoylethanolamide. Significant interest has recently also grown in therapies aiming at restoring the metabolic balance, improving microcirculatory flow, modulating key inflammatory mediators, and protecting cellular bioenergetics during severe infection. This review summarizes the main pathophysiological mechanisms involved in the sepsis-associated multiple organ dysfunction and discusses current and emerging pharmacological strategies in the field. By bringing these interconnected processes together, this review attempts to provide a clearer framework for our understanding of the disease progression and for the identification of more rational therapeutic targets. Such a framework can be of high importance, given that a stronger mechanistic understanding may help us improve treatment selection, optimize the timing of intervention(s), and support better clinical outcomes.

Keywords

mitochondrial dysfunction; multiple organ failure; oxidative stress; pharmacotherapy; sepsis

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PROCEEDINGS ABSTRACT

From inception to impact: Iraq's pharmacovigilance journey

Manal M. Younus

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Pharmacovigilance systems in low- and middle-income countries face significant structural, technical, and resource-related challenges that can limit their capacity to protect patients from medicine-related harm. Iraq, a country of approximately 46 million people located in Western Asia with a complex recent history, has established its national pharmacovigilance system relatively late compared to many regional counterparts: the Iraqi Pharmacovigilance Center (IPhvC) was founded on 31 December 2009 under the Ministry of Health's Directorate of Technical Affairs, with a vision of creating a safe medicinal environment and a mission centred on strengthening the detection, the assessment, the understanding, and the prevention of adverse drug reactions and medication errors. Iraq became a full member of the World Health Organization Program for International Drug Monitoring in November 2010. Over the subsequent 15 years, the IPhvC has pursued a sustained, multi-dimensional program of system development spanning ten interconnected areas. The database expansion progressed from a centralised national structure to a three-tiered network encompassing over 510 active VigiFlow (national database) accounts across regional centers, hospitals, specialized centers, and public health districts, including Kurdistan. The quantity of the reports grew substantially while the report quality (measured by VigiGrade completeness scores) consistently exceeded the global average of participating countries, reflecting the impact of structured training, mentoring, as well as the introduction of a dedicated pharmacovigilance unit manual. Furthermore, signal detection evolved from reactive case reviews to a more proactive approach using VigiLyze and disproportionality analysis, with the outputs feeding into regulatory action and safety communications. More importantly, data collection methodologies diversified beyond spontaneous reporting so as to include stimulated reporting introduced in 2019 and sentinel site active surveillance established in 2021, with the latter being applied in the case of the coronavirus disease 2019 (COVID-19) vaccine safety monitoring. The COVID-19 pandemic represented a critical stress test and accelerator for the system, prompting expanded reporting channels, decentralized surveillance structures, active membership in the expanded programme on immunization causality assessment committee, and the production of sixteen periodic safety reports shared with national regulatory and immunization bodies. In the meantime, marketing authorization holder pharmacovigilance has been progressively formalized (since 2012) through successive regulatory circulars, the introduction of national good pharmacovigilance practice guideline, requirements for pharmacovigilance system master files and periodic safety update reports, and the recent introduction of the industry e-reporting system that is aligned with the International Council for Harmonization E2B standard. Additional risk minimization measures grew from domestically identified safety signals, while a dedicated preventable medication errors project is nearing publication. Finally, substandard

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and falsified medicine surveillance, academic collaboration, MedSafetyWeek participation since 2018, the publication of a biannual pharmacovigilance bulletin, and the recent transfer of the national haemovigilance functions to the IPhvC further illustrate the breadth of the system's maturity development [1,2]. Despite this progress, challenges persist, including an uneven system maturity across stakeholders, a variable private-sector engagement, a limited patient involvement, and the need to link pharmacovigilance data with disease registries and health insurance systems. Overall, Iraq's experience demonstrates that a functional and improving national pharmacovigilance system can indeed be built within a complex environment through sustained political commitment, international partnerships, and institution-led capacity building.

Keywords

adverse drug reaction reporting; Iraq; medicine safety; national pharmacovigilance system; pharmacovigilance

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PROCEEDINGS ABSTRACT

Development, physicochemical characterisation, and antimicrobial activity assessment of selenium nanoparticles modified with chitosan and gallic acid

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Selenium (Se) nanoparticles (SeNPs) are widely considered as promising antimicrobial nanomaterials due to their biocompatibility, broad-spectrum biological activity, and relatively low toxicity compared to inorganic Se compounds. However, their poor stability and nanoparticle aggregation continue to be significant barriers to their use in biomedical applications. The aim of this study was to synthesise SeNPs that are stabilised and functionalised with chitosan and gallic acid, as well as to evaluate their physicochemical characteristics and antibacterial efficacy against *Staphylococcus aureus* and *Escherichia coli*. SeNPs were synthesised by chemical reduction using sodium selenite as the Se precursor and ascorbic acid as the reducing agent, in the presence of gallic acid and chitosan. The reaction mixture's obvious colour change from colourless to reddish–orange was the first indication of the nanoparticles' formation. Subsequently, Fourier-transform infrared (FTIR) spectroscopy, dynamic light scattering (DLS), zeta potential analysis, and scanning electron microscopy (SEM) were used in order to physiochemically characterise the produced SeNPs. Furthermore, the minimum inhibitory (MIC) and minimum bactericidal (MBC) concentrations against *S. aureus* and *E. coli* were calculated using the broth microdilution method in accordance with the 2020 CLSI M07 criteria. The undertaken FTIR analysis confirmed the successful functionalisation and stabilisation of the SeNPs by chitosan and gallic acid through hydrogen bonding and electrostatic interactions. The broad absorption band at 3331 cm^{-1} corresponded to overlapping O–H and N–H stretching vibrations, while the absorption band at 1637 cm^{-1} indicated interactions between the carbonyl and the deprotonated carboxylate groups of the gallic acid and the amino groups of chitosan. Additional absorption bands at the 1427–1316 cm^{-1} range corresponded to COO^- and C–N vibrations, whereas bands at the 1160–1035 cm^{-1} range confirmed the preservation of chitosan's backbone structure. The undertaken DLS analysis demonstrated that the average particle sizes of the chitosan-stabilised SeNPs (CS-SeNPs) and of the gallic acid-functionalised CS-SeNPs (GA/CS-SeNPs) were 142.0 and 137.6 nm, respectively. Following the addition of gallic acid, the polydispersity index values were found to be 0.241 and 0.209, thereby showing improved uniformity. The values of the undertaken zeta potential analysis were +38.3 mV for the CS-SeNPs and +38.5 mV for the GA/CS-SeNPs, indicating that these nanoparticles exhibit excellent colloidal stability. The creation of mostly spherical nanoparticles with particle sizes that are in line with the DLS findings was further verified by an SEM examination. Moreover, both nanoparticle formulations exhibited better antibacterial effectiveness against *S. aureus* than against *E. coli*, according to our *in vitro* studies. While the GA/CS-

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SeNPs exhibited increased antibacterial activity with MIC values down to 50 and 100 μg/mL against *S. aureus* and *E. coli*, respectively, the CS-SeNPs exhibited MIC values of 100 and 200 μg/mL against the same pathogens. At equal doses, gallic acid by itself did not stop bacterial growth, suggesting that the increased antibacterial action came from the functionalisation of the nanoparticles rather than from the free molecule. Moreover, according to our MBC analysis, the GA/CS-SeNPs exhibited values of 100 μg/mL against *S. aureus* and 200 μg/mL against *E. coli*, whereas the CS-SeNPs showed bactericidal action at 200 μg/mL against both bacterial strains. The developed GA/CS-SeNPs exhibit superior antibacterial activity, enhanced physicochemical stability, and improved nanoparticle uniformity, thereby indicating their potential as antimicrobial nanomaterials.

Keywords

antibacterials; chitosan; gallic acid; nanoparticles; selenium

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COLLOQUIA ERUDITA 2026; 1: 63–64

PROCEEDINGS ABSTRACT

Study of a novel series of compounds deriving from the 4-(4-chlorophenyl) cyclohexane-1-carboxylic acid: synthesis, spectroscopic characterization, and computational targeting of the CYP51 enzyme

Saja Alsalhy and Hussam W. Al-Humadi

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Heterocyclic compounds constitute a fundamental aspect of medicinal chemistry, with nitrogen-containing heterocycles being particularly important for drug discovery and development processes. Amongst these, the 1,2,4-triazole ring structure is a favoured framework due to the variety of its biological effects and its capacity to establish numerous non-covalent interactions with biological targets. The increasing occurrence of fungal infections and the development of resistance to current antifungal treatments highlight the critical demand for new therapeutic options. The aim of this study was to synthesise and to analyse a novel series of four 1,2,4-triazole derivatives (namely, compounds 5, 5a, 5b, and 5c), as well as to assess their antifungal activity potential. To this end, we have used molecular docking and *in silico* ADMET (absorption, distribution, metabolism, excretion, and toxicity) analysis in order to target the fungal cytochrome P450 14α-demethylase (CYP51). The synthesis began with the Fischer esterification of 4-(4-chlorophenyl) cyclohexane-1-carboxylic acid, followed by the formation of hydrazides, the preparation of the thiosemicarbazide intermediate compound 4, and its subsequent cyclisation leading to the production of the triazole core structure (compound 5). The last step included an *S*-alkylation with different alkylating agents so as to produce three unique derivatives (compounds 5a–5c). All compounds were analysed through the use of Fourier-transform infrared (FTIR) and proton nuclear magnetic resonance (^{1}H-NMR) spectroscopy, thereby verifying the successful creation of the intended structures with the identification of the distinctive spectral characteristics of the triazole ring system and of the added functional groups. The computational assessment of these new 1,2,4-triazole derivatives as prospective antifungal agents focused on the fungal CYP51; a critical target (enzyme) in the ergosterol biosynthesis process. Molecular docking analyses were undertaken on the fungal CYP51 in order to assess the binding affinities and modes of the four synthesized compounds. The docking findings have indicated that all four compounds can effectively fit into the enzyme's active site, forming multiple interactions with essential amino acid residues. The primary binding interactions comprised pi–alkyl, pi–sulphur, and alkyl interactions with important residues such as LYS_{151}, TYR_{140}, HIS_{468}, PHE_{463}, and CYS_{470}. Moreover, the binding affinities of the newly synthesized compounds were found to be similar to that of fluconazole (i.e., the reference antifungal medi-

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cation), thereby suggesting a potential antifungal effect through the inhibition of CYP51. Compound 5c exhibited particularly notable binding properties, displaying extensive interactions within the CYP51 active site with a binding affinity of -10.1 kcal/mol. *In silico* ADMET assessments were performed through the use of the SwissADME tool in order to assess the drug-likeness and the pharmacokinetic properties of the synthesized compounds. All of the herein assessed compounds adhered to Lipinski's rule of five, thereby indicating that they possess favourable physicochemical traits for oral bioavailability. In fact, the compounds exhibited suitable topological polar surface area values and high bioavailability scores, revealing a promising potential for oral absorption. Interestingly, while most compounds were expected to be found to be suitable substrates for P-glycoprotein efflux transporters with generally low gastrointestinal absorption, compound 5 exhibited high gastrointestinal absorption. Finally, none of the herein assessed compounds is expected to be able to penetrate the blood–brain barrier, suggesting a lower risk of central nervous system-associated side effects. The promise of these novel 1,2,4-triazole derivatives as viable candidates for antifungal drug development is supported by the aforementioned computational findings; however, the undertaking of *in vitro* and *in vivo* studies is required in order to confirm their therapeutic efficacy and safety profiles.

Keywords

antifungal agents; CYP51 inhibition; heterocyclic compounds; Lipinski's rule of five; SwissADME

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PROCEEDINGS ABSTRACT

Critical serum cytokine levels of rats subjected to high-fat diet- and letrozole-induced experimental model of the polycystic ovary syndrome: modulation by dietary probiotic supplementation

Bassam Hasan, Aymen A. Bash, and Lena Fadhil Hamza

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The polycystic ovary syndrome (PCOS) is an endocrine and metabolic disease, and a major cause of anovulatory infertility in women worldwide. The most commonly used drug for the treatment of PCOS is metformin, which enhances insulin sensitivity and glucose uptake into the peripheral tissue, and decreases glucose production by the liver. According to recent research, microbial imbalances may increase intestinal permeability resulting in the release of endotoxins into the bloodstream; these endotoxins can trigger low-grade systemic inflammation with high levels of tumor necrosis factor alpha (TNF-α) and low levels of inerleukin-10 (IL-10), thereby contributing to the development of insulin resistance and ovarian dysfunction. The aim of this study was to determine whether a dietary supplementation with probiotics could reduce the inflammatory response (by restoring the serum levels of TNF-α and IL-10 to normal ones) in a high-fat diet- and letrozole-induced model of PCOS in rats. To this end, a total of 28 female Wistar rats were randomly assigned into four groups (n=7 each): a control group, a PCOS (positive control) group, a metformin-treated PCOS group, and a probiotic-receiving PCOS group. Letrozole (an aromatase inhibitor) was administered orally (*via* gavage) at a dose of 1 mg/kg/day along with a high-fat diet (with a total kcal value of 4.73 kcal/g) that was obtained from Research Diets, Inc. (USA). The PCOS-undergoing rats were returned into the same diet as the control group after 21 days of stimulation and the letrozole administration was then also stopped. The metformin-treated PCOS group was treated with 300 mg/kg/day of metformin in distilled water, while the rats in the probiotic-receiving PCOS group were fed with commercially available probiotics obtained from Medwise Healthcare Limited (UK), at a dose of 1×10^9 CFU/day for 28 days. The measurement of the serum IL-10 and TNF-α levels was performed after 28 days of treatment. Our findings revealed that the experimental simulation of PCOS in rats significantly increased the TNF-α levels by 46.33% ($p<0.001$) and significantly decreased those of IL-10 by 32.98% ($p<0.001$) compared to the respective TNF-α and IL-10 levels of the control group, thereby highlighting the existence of low-grade systemic inflammation in the PCOS group. In the rats of the metformin-treated PCOS group, a significant increase in their IL-10 levels (+36.88%; $p<0.001$) and a moderate decrease in their TNF-α levels (-7.71%; $p>0.05$) were observed as compared to those of the PCOS group. The serum levels of

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IL-10 were also found to be significantly increased by 27.51% (p<0.001) in the probiotic-receiving PCOS group when compared with those of the PCOS group, and significantly decreased by 14.54% (p=0.003) when compared with those of the control group. Finally, the serum TNF-α levels were found to be slightly decreased by 4.69% (p>0.05) in the rats of the probiotic-receiving PCOS group when compared with those of the PCOS group, and significantly increased by 39.46% (p=0.001) when compared with those of the control group. Therefore, our findings suggest that a supplementation with probiotics is not enough in order to achieve normal serum IL-10 and TNF-α levels in an experimental model of PCOS in rats.

Keywords

inflammation; letrozole; metformin; PCOS; probiotics

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PROCEEDINGS ABSTRACT

The modulatory role of semaglutide on nesfatin-1 in metabolic disorder: a review

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The dysregulation of neuroendocrine circuits that regulate appetite, glucose metabolism, and energy balance leads to metabolic diseases such as obesity and type 2 diabetes mellitus. Semaglutide is a long-acting glucagon-like peptide-1 (GLP-1) receptor agonist capable of enhancing glycaemic management and weight loss by stimulating glucose-dependent insulin secretion, suppressing glucagon release, delaying stomach emptying, and activating central satiety pathways. Moreover, it is known to promote weight loss by suppressing appetite through the hypothalamic GLP-1 receptors, and it affects the brainstem and the hypothalamus; two parts of the brain that regulate satiety. Semaglutide decreases food cravings and caloric intake, while weight loss improves metabolic parameters such as insulin sensitivity and lipid profile. Through weight loss and increased insulin sensitivity, semaglutide improves aspects of the metabolic syndrome, such as central obesity, hyperglycaemia, dyslipidaemia, hypertension, and cardiometabolic disease. On the other hand, nesfatin-1 is an anorexigenic peptide (found in both central and peripheral tissues) that has a role in thermogenesis, glucose homeostasis, lipid metabolism, hunger control, and stress reactions. Its capacity to reduce food intake results from its ability to suppress hunger-promoting neurons and to activate hypothalamic satiety centers in hypothalamic satiety signalling. Nesfatin-1 can act in multiple ways in order to prevent hyperglycaemia. Due to the fact that nesfatin-1 and insulin are co-localized in the β-cells of the pancreatic islets of Langerhans, when blood glucose levels rise, insulin and nesfatin-1 are co-released. In skeletal muscle and adipose tissues, it increases the translocation of the glucose transporter type 4 to the plasma membrane, enabling cells to more effectively remove glucose from the bloodstream. Furthermore, nesfatin-1 can directly inhibit glucagon secretion under hyperglycaemic conditions. It is also known to stimulate sympathetic nervous system outflow, brown adipose tissue thermogenesis, metabolic rate, and heat production, all of which enhance whole-body energy expenditure. Consequently, reduced body weight and negative energy balance are encouraged by this combination of reduced intake and increased expenditure. Peripherally, nesfatin-1 can directly affect the hepatocytes and the adipocytes by downregulating lipogenic factors and by upregulating critical lipolytic enzymes; increased levels of fatty acid oxidation, decreased triglyceride buildup, and improved blood lipid profiles are some of the results of these actions, which have a preventive role against metabolic syndrome and obesity. According to experimental studies, nesfatin-1 seems to increase the intestinal L-cells' production of GLP-1, thereby indicating its potential upstream function in enhancing the effects of the GLP-1-mediated pathway. On the other hand, it has been shown that a semaglutide treatment can increase nesfatin-1 expression in both central and peripheral tissues, suggesting the existence of a positive feedback mechanism between the

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endogenous nesfatin-1-mediated satiety and the GLP-1 receptor activation. Overall, semaglutide and nesfatin-1 seem to function similarly by reducing stomach emptying, suppressing food intake, increasing insulin secretion, and inhibiting glucagon release. These findings emphasize the overlap of the gut–brain and the neuroendocrine pathways in the control of energy balance. The strong appetite suppression and weight loss seen with GLP-1 receptor agonist therapy may be explained mechanistically by nesfatin-1 acting as an endogenous mediator and an amplifier of semaglutide's actions. In conclusion, semaglutide and nesfatin-1 are correlated, indicating the existence of a synergistic interaction between endogenous anorexigenic pathways and drug-induced GLP-1 receptor activation. There is evidence that the GLP-1 signalling and the nesfatin-1 pathways interact functionally, as the systems have similar mechanisms and semaglutide can increase nesfatin-1 expression. In addition to highlighting nesfatin-1 as a possible biomarker and treatment target for obesity and type 2 diabetes mellitus, this review suggests that an understanding of this interplay may offer fresh perspectives on metabolic regulation.

Keywords

GLP-1; nesfatin-1; obesity; semaglutide; type 2 diabetes mellitus

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PROCEEDINGS ABSTRACT

The roles of liraglutide and visfatin in metabolic and inflammatory regulation: a review

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Liraglutide is a glucagon-like peptide-1 (GLP-1) receptor agonist that is frequently used for the management of obesity and type 2 diabetes mellitus. Liraglutide attaches to GLP-1 receptors located on pancreatic β-cells, leading to the promotion of insulin release exclusively when blood sugar levels are elevated. At the same time, it suppresses glucagon secretion from α-cells, thereby decreasing blood glucose levels. Additionally, the medication reaches the appetite regulation centers (specifically, the hypothalamus and the brainstem), enhances feelings of fullness and diminishes hunger, resulting in a reduction of energy intake. Furthermore, liraglutide delays gastric emptying, which in turn slows the absorption of nutrients, including glucose, into the bloodstream and mitigates postprandial blood sugar surges. Its impact on inflammatory pathways, adipokine modulation, and the biology of adipose tissue is given special consideration in this review. The adipokine visfatin, which is mostly generated in visceral adipose tissue, is crucial for energy balance, inflammatory signalling, and metabolic control. This review outlines the functions of both intracellular and extracellular visfatin in NAD metabolism, mitochondrial function, and systemic inflammation, and focuses on the possible relationship between liraglutide administration and circulating visfatin levels through enhanced adipose tissue function, weight loss, and the suppression of inflammatory pathways. Liraglutide-induced improvements in adipose tissue physiology may affect circulating visfatin levels because the latter is primarily released by visceral adipose tissue and is linked to metabolic stress and inflammation. Visceral adiposity reduction is one of the main pathways connecting liraglutide to visfatin. In metabolic disorders, visceral adipose tissue is considered as a key source of circulating visfatin. Liraglutide therapy suppresses appetite and modifies central satiety pathways so as to promote weight loss and to decrease the visceral fat mass. Visfatin (as well as other pro-inflammatory adipokines) may undergo decreased secretion when the adipocyte hypertrophy and the adipose tissue inflammation are reduced. Liraglutide affects inflammatory pathways in the adipose tissue in addition to body weight and adiposity. Increased activation of inflammatory signalling pathways, including those of the nuclear factor kappa-light-chain-enhancer of activated B cells and the mitogen-activated protein kinase, can promote the synthesis of cytokines (such as tumor necrosis factor-α, interleukin-1β, and interleukin-6) and is a hallmark of chronic metabolic diseases. It has been observed that visfatin can amplify inflammatory signals in the adipose tissue. Liraglutide may indirectly lessen inflammatory signals linked to high visfatin levels, by inhibiting the generation of inflammatory cytokines and by promoting anti-inflammatory macrophage phenotypes in the adipose tissue. In conclusion, there might be a crucial connection between pharmacological GLP-1 receptor activation and adipokine regulation through the interaction between li-

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raglutide and visfatin. By reducing visceral adiposity, by improving metabolic homeostasis, and by suppressing inflammatory signalling within the adipose tissue, liraglutide may indirectly influence visfatin expression and its circulating levels. Our review also suggests that visfatin should be further explored as a biomarker for treatment responsiveness in obesity and type 2 diabetes mellitus.

Keywords

diabetes; inflammation; liraglutide; obesity; visfatin

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PROCEEDINGS ABSTRACT

Immunosenescence and inflammaging: molecular mechanisms, age-related diseases, and therapeutic interventions

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Aging is a complex biological process marked by progressive cellular and molecular changes that affect all physiological processes, especially those related to the immune system. Amongst these, increased susceptibility to infections, decreased vaccine efficacy, and a higher incidence of chronic diseases are all consequences of immunosenescence. The latter is defined as "the age-related decline and dysregulation of immune responses". The tissue accumulation of senescent cells that release a variety of pro-inflammatory mediators is collectively referred to as the senescence-associated secretory phenotype (SASP) and is known to be capable of causing chronic low-grade inflammation / "inflammaging". Inflammaging is a key component of the process that accelerates immune cell aging, reduces the efficient clearance of senescent cells, and limits the immune response to novel antigens. This decline of both innate and adaptive immunity is significantly influenced by this ongoing inflammatory state, resulting in delayed immune responses and compromised host defence systems. This review provides an overview of the molecular drivers of immunosenescence and inflammaging, and discusses their roles in age-related diseases and their importance for targeted therapeutic interventions. Immunosenescence is controlled at the molecular level by a number of interrelated molecular signalling pathways. Amongst these, the nuclear factor kappa B (NF-κB) pathway is a major cause of chronic inflammation because it inhibits the senescent cells' ability to undergo apoptosis and stimulates the production of immune mediators. Similarity, immunological dysfunction, and decreased T lymphocyte responsiveness are caused by a dysregulation of the mammalian target of rapamycin (mTOR) pathway, which also affects autophagy and cellular metabolism. Furthermore, by detecting cytosolic DNA and by inducing type I interferons, the activation of the cyclic GMP–AMP synthase / stimulator of interferon genes (cGAS-STING) pathway has become a significant mediator of age-related inflammation. These inflammatory signalling pathways are further amplified by oxidative stress and mitochondrial dysfunction. Significant cellular changes are linked to immunosenescence and include telomere attrition, thymic involution, haematopoietic stem cell dysfunction, and reduced naïve T lymphocyte production; all these impair immune surveillance and response to novel antigens. Reduced B lymphocyte diversity and antibody production are also consequences of this process. Together, these alterations play a key role in the pathophysiology of age-related diseases (such as cancer), severe viral infections (such as the coronavirus disease 2019), cardiovascular diseases, neurodegenerative diseases (such as Alzheimer's disease), and type 2 diabetes mellitus. In fact, immune dysregulation and chronic inflammation

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serve as common underlying mechanisms connecting immunosenescence to the aforementioned pathologies. Recent approaches in the field of therapeutics have been targeting the underlying mechanisms of immune aging. Senotherapeutics, being the use of senomorphics in order to alter the secretory phenotype of senescent cells and senolytics that specifically eradicate them, have demonstrated encouraging outcomes in both preclinical and clinical studies. Through the modification of important pathways such as the mTOR and NF-κB ones, pharmaceutical agents like metformin and rapamycin can lower inflammation and boost immunity. Cytokine modulation, immune-based treatments, and lifestyle modifications (including the adoption of regular physical activity, balanced nutrition, and adequate sleep) have all been shown to exert positive effects on immune aging. In conclusion, immunosenescence is a multifaceted process that is forced by intricate molecular pathways and cellular changes, and that has important ramifications for the emergence of age-related diseases. However, the development of targeted therapeutic interventions that are meant to improve immune function, increase longevity, and encourage healthy aging, requires a deeper understanding of these mechanisms and their clinical implications.

Keywords

aging; immunosenescence; inflammaging; senotherapeutics; signalling pathways

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PROCEEDINGS ABSTRACT

Recent advances in the design and synthesis of vascular endothelial growth factor receptor 2 antagonists: a review

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Angiogenesis, the process by which new blood vessels emerge from existing ones, is a tightly regulated physiological process vital to growth, tissue regeneration, and homeostasis. Abnormal angiogenesis is the feature of many pathologies and especially of cancer, where it facilitates the growth of tumours, their invasion, and their metastasis. The vascular endothelial growth factor (VEGF) signalling pathway is central to the regulation of angiogenesis, while the vascular endothelial growth factor receptor 2 (VEGFR-2) is the primary mediator of VEGF-induced endothelial activities. VEGFR-2 has become a key therapeutic target in cancer treatment due to its central role in promoting endothelial cell proliferation, migration, survival, and vascular permeability. The aim of this review was to critically assess recent developments in the design and synthesis of VEGFR-2 antagonists, with a focus on innovative small-molecule inhibitors, computational drug design methodologies, macromolecular therapeutic strategies, and emerging approaches to overcome resistance and toxicity associated with anti-angiogenic therapy. VEGFR-2 is a receptor tyrosine kinase with its structure consisting of an extracellular ligand-binding domain, a transmembrane domain, and an intracellular tyrosine kinase domain. VEGFR-2 stimulation through the VEGF binding induces receptor dimerization and autophosphorylation, thereby promoting angiogenesis *via* downstream signalling pathways. Detailed knowledge of the VEGFR-2 structure, particularly of its ATP-binding site, its hinge region, its hydrophobic pockets, and its activation loop, has enabled the rational design of selective VEGFR-2 inhibitors targeting its kinase activity. Recent progress in medicinal chemistry has led to the emergence of various classes of VEGFR-2 inhibitors that are more potent and selective. Amongst these, nicotinamide-thiadiazol hybrids exhibit high inhibitory activity and excellent anticancer capacity due to their effective interactions with the ATP-binding domain. Benzofuran derivatives, including clinically used agents such as sorafenib, have exhibited greater selectivity and cytotoxicity against hepatocellular carcinoma. On the other hand, peptide-tetrahydroisoquinoline conjugates represent an emerging design strategy to enhance solubility, target specificity, and pharmacokinetic properties through a hybrid molecular design. Urea-functionalized 2-oxoindolin-3-ylidene analogues have exhibited pronounced antiproliferative effects through the formation of essential hydrogen bonds with the kinase and the stabilization of inactive forms of VEGFR-2. Computational approaches have also been utilised, thereby enabling the discovery of VEGFR-2 inhibitors to proceed even faster: amongst these, the employment of three-dimensional quantitative structure–activity relationship modelling, molecular docking, and molecular dynamics simulations have

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allowed for accurate predictions of the ligand–receptor interactions, optimization of binding affinity, and determination of complex stability. Virtual screening and chemical space exploration have also increased the discovery of new scaffolds beyond the traditional kinase inhibitor chemotypes, thereby helping overcome the shortcomings of resistance and structural redundancy. In addition to small molecules, novel therapeutic platforms are currently being explored, including peptide–drug conjugates and bispecific antibody-drug conjugates, such as JSKN027, that combine VEGFR-2 targeting with immune checkpoint inhibition (e.g., PD-L1). These multipurpose systems could provide synergy by combining the anti-angiogenic effects with immunomodulatory and targeted cytotoxicity. Nonetheless, the acquired resistance, off-target toxicity, and lack of selectivity remain challenges despite the aforementioned tremendous pharmacological advancements. Combination therapies, biomarker-directed therapy, enhanced targeting specificity, and sophisticated drug delivery systems are among the strategies that can be used in order to overcome these problems. Moreover, recent advances in structure-based drug design, *in silico* approaches, and multifunctional therapeutics are radically reshaping the VEGFR-2-targeted therapeutics. Finally, the anti-angiogenic therapy for cancer is expected to improve in the future through the use of artificial intelligence and precision medicine approaches, thereby increasing both treatment safety and clinical efficacy.

Keywords

angiogenesis; *in silico* drug design; peptide–drug conjugates; therapeutics; VEGFR-2

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PROCEEDINGS ABSTRACT

The utility, effectiveness, and challenges of the use of antibody–drug conjugates in targeted cancer therapy: a narrative review

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Cancer remains one of the leading causes of morbidity and mortality worldwide, with approximately 20 million new cases and 9.7 million deaths reported in 2022. Global projections indicate that the annual incidence of cancer could exceed 35 million cases by 2050 due to population growth, ageing, and increasing exposure to risk factors such as smoking and obesity. Although surgery, radiotherapy, and conventional chemotherapy remain fundamental treatment modalities, their clinical utility is often limited by inadequate tumour selectivity, systemic toxicity, and the emergence of drug resistance. The aim of this narrative review was to summarise the utility, therapeutic effectiveness, and current challenges associated with antibody–drug conjugates (ADCs) in targeted cancer therapy, while highlighting recent innovations that may enhance their clinical impact. ADCs represent an important advancement in precision oncology by combining the high specificity of monoclonal antibodies with the cytotoxicity of anticancer agents. These agents function through selective binding to tumour-associated antigens expressed on malignant cells, followed by the receptor-mediated internalization and intracellular release of payloads. This targeted delivery strategy allows ADCs to maximize antitumour activity while minimizing exposure of healthy tissues. ADCs frequently employ humanised IgG1 antibody backbones, providing prolonged circulation times and additional immune-mediated mechanisms such as antibody-dependent cytotoxicity and complement activation. Clinical effectiveness of ADCs has been demonstrated across a growing range of haematological malignancies and solid tumours. By late 2024, approximately 15 ADCs had received approval from the United States FDA for various oncological indications. Notable examples include gemtuzumab ozogamicin for acute myeloid leukaemia and trastuzumab deruxtecan for human epidermal growth factor receptor 2 (HER2)-positive and HER2-low breast cancer. These successes have established ADCs as a major therapeutic platform. Despite their promise, several challenges continue to limit the broader application of ADCs: drug delivery efficiency remains relatively low, with only a small proportion of the administered dose reaching tumour tissues, while clinically significant toxicities (including interstitial lung disease, neutropenia, and ocular adverse effects) may compromise the treatment outcomes and contribute to trial discontinuation. Additional barriers include complex manufacturing processes, high production costs, and limited accessibility in resource-constrained settings. Moreover, the emergence of acquired resistance through antigen downregulation, epitope alterations, and enhanced drug efflux mechanisms further complicates long-term treatment success. In order to overcome these limitations, next-generation

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ADC technologies are being actively developed. These include bispecific ADCs capable of targeting multiple antigens simultaneously, probody–drug conjugates that are designed for selective activation within the tumour microenvironment, degrader–antibody conjugates utilising targeted protein degradation strategies, and immune-stimulating ADCs enhancing antitumor immune responses. These innovations, together with advances in site-specific conjugation and combination therapies involving immune checkpoint inhibitors, are expected to improve the safety, efficacy, and clinical utility of ADCs. The latter are, therefore, likely to remain central to the continuing evolution of personalised and targeted cancer treatment.

Keywords

antibody–drug conjugates; cancer therapy; monoclonal antibodies; precision medicine; targeted drug delivery

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PROCEEDINGS ABSTRACT

Molecular basis of drug-induced organ toxicity and advances in protective pharmacological interventions: a review

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Drug-induced toxicity is a major clinical and pharmacological challenge that continues to contribute significantly to patient morbidity, prolonged hospitalisation, and drug withdrawal worldwide. Although drugs are developed with the aim to provide therapeutic benefits, their biochemical and molecular interactions within the body are complex and may lead to unintended harmful effects involving multiple organ systems. Such adverse effects can arise through several mechanisms of toxicity, involving (but not limited to) dose-dependent toxicity, immune-mediated toxicity, mitochondrial dysfunction and oxidative stress, disruption of cellular homeostasis, and the activation of inflammatory or apoptotic signalling pathways; these processes may be occurring individually or may be synergistically leading to tissue injury and organ dysfunction. In addition, interindividual variability (particularly in terms of genetic polymorphisms affecting drug metabolism and transport, as well as detoxification pathways) can play a significant role in determining drug disposition and susceptibility to toxicity, making some patients more vulnerable than others even at standard therapeutic doses. In addition, environmental factors, drug interactions, and underlying comorbidities may further increase the risk and severity of toxic responses and drug adverse reactions. Among the organs / systems most frequently affected by drug-induced toxicity are the liver, the kidneys, the heart, and the auditory system. Hepatotoxicity is commonly associated with the cytochrome P450-mediated metabolism of drugs into reactive intermediates, which promote oxidative stress, the depletion of intracellular antioxidants such as glutathione, and the subsequent hepatocellular injury / necrosis. Nephrotoxicity often results from mitochondrial impairment, inflammatory responses, oxidative damage, as well as direct tubular cell damage, particularly with drugs such as aminoglycosides and cisplatin. Cardiotoxicity, especially the one induced by anthracyclines such as doxorubicin, is linked to multiple mechanisms including topoisomerase inhibition, iron dysregulation, calcium imbalance, excessive reactive oxygen species generation, and mitochondrial dysfunction, ultimately leading to myocardial injury and impaired cardiac function. Finally, ototoxicity, which is frequently associated with aminoglycosides and platinum-based anticancer drugs, involves a disruption of ion homeostasis, the excessive production of reactive oxygen species, and an irreversible damage to cochlear hair cells resulting in hearing impairment or permanent hearing loss. The better understanding of the molecular mechanisms involved in such cases of drug-induced organ injury has contributed to the development of several protective pharmacological strategies aimed at minimizing toxicity and improving patient safety. These strategies

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involve the use of antioxidant agents, enzyme modulators, mitochondrial protectants, transporter inhibitors, and immunomodulators. A common example of such strategies is the use of *N*-acetylcysteine in counteracting acetaminophen-induced hepatotoxicity by restoring intracellular glutathione levels. Dexrazoxane is also commonly employed for the reduction of the anthracycline-induced cardiotoxicity. Other approaches aim at the modulation of signalling pathways, the enhancement of endogenous antioxidant defences, the reduction of inflammatory responses, and the stabilization of mitochondrial integrity and function. The further development of pharmacogenomics, the establishment of nanotechnology-based drug delivery systems, and the launch of reliable artificial intelligence-driven toxicity prediction platforms are all gaining increasing attention for their potential to enhance drug safety and support personalised therapeutic strategies and precision medicine. These innovations may allow for the early identification of high-risk individuals, the better monitoring of drug responses, and a more precise optimization of dosing regimens in clinical practice. This review provides an overview of key molecular mechanisms underlying drug-induced organ toxicity and highlights established and emerging protective pharmacological strategies. A deeper understanding of these interconnected processes may support safer drug development, improve the early detection of adverse effects, and contribute to the development of more individualised treatment approaches, thereby enhancing clinical outcomes and minimizing drug-induced harm in diverse patient populations.

Keywords

cardiotoxicity; drug toxicity; hepatotoxicity; nephrotoxicity; protective strategies

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PROCEEDINGS ABSTRACT

Novel thiadiazole–thiourea hybrids targeting the vascular endothelial growth factor receptor 2: design, synthesis, characterization, and *in silico* assessment

Noora A. Al-Jubouri, Shaker A. Abdul Hussein, and Hussam W. Al-Humadi

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A novel series of 1,3,4-thiadiazole-thiourea hybrid derivatives (namely, A-Cl, B-NO_2, C-OH, D-CH_3, and E-OCH_3) was systematically conceived, synthesized, and assessed for their efficacy as inhibitors of the vascular endothelial growth factor receptor 2 (VEGFR-2), thereby aimed at a critical mediator of tumour angiogenesis. The synthetic methodology adopted a convergent two-step strategy. Initially, the key intermediate, 5-amino-1,3,4-thiadiazole-2-thiol, was subjected to *S*-alkylation with a variety of substituted phenacyl bromides (bearing Cl, NO_2, OH, CH_3, and OCH_3) in acetone, at room temperature, affording the corresponding *S*-alkylated intermediates (namely, A–E). Subsequently, the target compounds were proficiently synthesized through the introduction of the thiourea moiety by reacting the amino group of each *S*-alkylated intermediate with 1-isothiocyanato-4-methoxybenzene in acetonitrile under reflux conditions for 24 h. Utilising Fourier transform infrared spectroscopy (FTIR) in conjunction with proton nuclear magnetic resonance (^{1}H-NMR) and carbon-13 nuclear magnetic resonance (^{13}C-NMR) has led to an effective validation of the chemical structures. Subsequently, by using the MOE 2024.06 software, we carried out molecular docking investigations, centring on the VEGFR-2 kinase domain (PDB ID: 4ASD), with sorafenib serving as the reference. The method of docking was confirmed through the effective re-docking of the co-crystallized ligand, resulting in a root mean square deviation (RMSD) measurement of 1.60 Å. Each derivative that was synthesized displayed encouraging binding affinities, with docking scores ranging from -7.94 to -8.64 kcal/mol, which were found to be similar to that of sorafenib (-9.23 kcal/mol). Interestingly, the nitro-substituted derivative (B-NO_2) has risen to the forefront as the most captivating compound, exhibiting a binding affinity (-8.64 kcal/mol) that approached much that of sorafenib. This increased affinity was ascribed to an optimal amalgamation of conserved hydrogen bonding interactions with the critical active site residues ASP_{1046}, LYS_{868}, and GLY_{922}, further augmented by hydrophobic interactions with LEU_{889}, LEU_{1035}, LEU_{840}, ALA_{866}, ILE_{892}, VAL_{899}, VAL_{916}, CYS_{919}, LEU_{1019}, VAL_{898}, and ILE_{1044}. Additionally, the B-NO_2 compound maintained a significant pi–sulfur interaction with CYS_{1045} and a salt bridge interaction with GLU_{885}, further enhancing its binding stability. By using SwissADME for *in silico* ADME (absorption, distribution, metabolism, and excretion) profiling, it was shown that every compound synthesized followed the Lipinski rule of five with no violations, thereby strengthening their qualities as potential drugs. The derivatives exhibited advantageous physicochemical features, comprising acceptable molecular weights

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ranging from 430.57 to 461.54 g/mol, hydrogen bond donor numbers of 2 to 3, and acceptor values from 4 to 6, plus a moderate to high lipophilicity reflected in a consensus log P of 2.79 to 4.12. While all compounds were shown to possess a low gastrointestinal absorption resulting from their relatively elevated topological polar surface area (TPSA; being >161.77 $Å^2$), they exhibited satisfactory bioavailability readings (0.55). More importantly, none of these derivatives exhibited potential PAINS (pan-assay interference compounds) alerts and none are anticipated to penetrate the blood–brain barrier, which is advantageous for minimising the risk of triggering adverse effects associated with the central nervous system. However, the profiling of cytochrome P450 inhibition has suggested possible inhibitions of CYP2C19, CYP2C9, and CYP3A4 from all compounds, while the B-NO_2 and E-OCH_3 ones exerted no inhibition of CYP1A2, thereby emphasizing the necessity for undertaking an assessment of potential drug–drug interactions. In conclusion, we have herein successfully devised and synthesized a novel series of thiadiazole–thiourea derivatives with validated structural integrity; the nitro-substituted derivative B-NO_2 has emerged as a promising candidate for VEGFR-2 inhibition, exhibiting superior binding affinity through optimal molecular interactions within the kinase active site as well as favourable drug-like properties *in silico*, thereby establishing a foundation for its further optimization and development as an anti-angiogenic drug.

Keywords

anticancer; molecular docking; 1,3,4-thiadiazole; thiourea; VEGFR-2

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PROCEEDINGS ABSTRACT

Nanocomposite-based drug delivery systems for targeted anti-inflammatory therapies: a review

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Inflammatory diseases remain a clinical challenge as persistent or dysregulated inflammation contributes to tissue injury and the progression of acute and chronic disorders. Conventional anti-inflammatory therapies, including those using nonsteroidal anti-inflammatory drugs and glucocorticoids, are often limited by systemic toxicity, poor tissue specificity, short biological half-life, and inadequate control of complex inflammatory pathways. However, nanocomposite-based drug delivery systems, particularly polymeric nanoparticle platforms, have emerged as promising strategies to overcome these limitations through targeted, sustained, and disease-responsive drug delivery. The aim of this review was to provide an overview of the role of polymer-based nanocomposite delivery systems in targeted anti-inflammatory therapies, with an emphasis on the influence of polymer composition, functional properties, and disease-specific applications on the therapeutic outcomes. Polymeric nanoparticles are nanoscale carriers fabricated from synthetic or natural polymers, including poly(lactic-co-glycolic acid), chitosan, and polyethylene glycol (PEG). These materials enable the efficient encapsulation and controlled release of anti-inflammatory agents while providing advantages such as biodegradability, biocompatibility, prolonged circulation time, and reduced immune clearance. The physicochemical properties of the selected polymer strongly influence drug-loading capacity, biodistribution, pharmacokinetics, cellular uptake, and overall therapeutic efficacy. Furthermore, hybrid nanocomposite systems that combine synthetic and natural polymers offer enhanced versatility by integrating the favourable characteristics of multiple materials into a single platform. The therapeutic relevance of polymer-based nanocomposites is closely linked to their application across diverse inflammatory disorders. In autoimmune diseases, such as rheumatoid arthritis and inflammatory bowel disease, these systems can facilitate targeted delivery of anti-inflammatory drugs to inflamed tissues and immune cells, thereby improving local therapeutic efficacy while minimizing systemic adverse effects. In acute inflammatory conditions, nanocomposite carriers can be engineered to respond to pathological stimuli, including elevated reactive oxygen species levels, acidic pH, hypoxia, and enzyme overexpression, thereby enabling localized drug release at the inflammation sites. In neuroinflammatory disorders, polymeric nanocarriers offer additional advantages by enhancing transport across the blood–brain barrier, reducing microglial activation, modulating inflammatory cytokine production, and promoting neuronal protection. Beyond functioning as passive drug carriers, polymeric nanocomposites can actively modulate inflammatory pathways. Experimental studies have demonstrated their ability to promote macrophage phenotype reprogramming, suppress excessive cytokine production, inhibit nuclear factor kappa B signalling, and scavenge reactive oxygen species. Surface functionalization with PEG, target-

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ing ligands, peptides, or antibodies can further enhance the selective accumulation within inflamed tissues and activated immune cell populations. In addition, stimuli-responsive polymer designs can provide precise spatial and temporal control of drug release within diseased microenvironments, thereby supporting the development of personalised therapeutic approaches. Overall, the success of any nanocomposite-based anti-inflammatory therapy depends largely on the selection of polymer type and its functional characteristics. Synthetic and natural polymers can each offer distinct advantages, and their rational design has expanded therapeutic opportunities in autoimmune, acute, and neuroinflammatory diseases. Although challenges related to large-scale manufacturing, reproducibility, regulatory approval, and clinical translation remain, polymer-based nanocomposite delivery systems represent a promising approach for improving the safety, the specificity, and the effectiveness of anti-inflammatory treatments.

Keywords

drug delivery; inflammation; nanocomposite systems; polymeric nanoparticles; targeted therapy

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COLLOQUIA ERUDITA 2026; 1: 83–84

PROCEEDINGS ABSTRACT

Protective effects of a *Tephrosia purpurea* extract, alone and in combination with *N*-acetylcysteine, on the hepatic injury and critical liver function markers of rats exposed to carbon tetrachloride

Huda I. Al-Khafaji and Qayssar Joudah Fadheel

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The carbon tetrachloride (CCl_4)-induced hepatotoxicity is widely used as an *in vivo* experimental model for simulating liver injuries that are associated with oxidative stress, hepatocellular degeneration, and impaired hepatic function. *Tephrosia purpurea* is a flowering plant species that possesses antioxidant and hepatoprotective properties, whereas *N*-acetylcysteine (NAC) is a well-established antioxidant that can replenish intracellular glutathione stores and limit oxidative tissue damage. This study evaluated the protective effects of a *Tephrosia purpurea* extract, administered alone or in combination with NAC, against CCl_4-induced liver injury in Wistar rats. To this end, 48 adult male Wistar rats were randomly allocated into eight groups (n=6 per group): (i) a normal control group, (ii) a negative control (olive oil) group, (iii) a positive control (CCl_4-treated) group, (iv) a group receiving NAC (at 150 mg/kg) prior to the CCl_4 administration, (v–vii) three groups receiving *Tephrosia purpurea* extract (at doses of 100, 200, and 400 mg/kg, respectively) prior to CCl_4 administration, and (viii) a combination group receiving NAC (at 150 mg/kg) together with a *Tephrosia purpurea* extract (at 200 mg/kg) before the CCl_4 administration. Treatments were administered orally, once daily, for 28 days, while hepatotoxicity was induced by the intraperitoneal administration of CCl_4 (1 mL/kg; 1:1 in olive oil) twice weekly. Hepatoprotection was assessed through histopathological examination of the liver and through the measurement of liver function biomarkers. The undertaken histopathological examination demonstrated normal hepatic architecture in both the normal and negative control groups, whereas the CCl_4-treated (positive control) group exhibited marked hepatocellular degeneration, necrosis, inflammatory cell infiltration, and architectural distortion. Pretreatment with NAC and *Tephrosia purpurea* extract significantly reduced the histological injury score when compared with that of the CCl_4-treated (positive control) group ($p<0.001$). The extract of *Tephrosia purpurea* exerted a dose-dependent improvement, reducing the histological injury score by 45.3%, 54.1%, and 88.8% at doses of 100, 200, and 400 mg/kg, respectively ($p<0.001$). The combination of NAC with the *Tephrosia purpurea* extract has produced the greatest hepatoprotective effect, reducing the histological injury score by 91.2% when compared with that of the CCl_4-treated (positive control) group ($p<0.001$). The undertaken biochemical analysis demonstrated no significant differences between the normal and negative control

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groups (p>0.05). In contrast, the CCl_4-treated (positive control) group exhibited significant liver dysfunction, characterized by increased serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and total bilirubin levels, along with decreased serum albumin and total protein concentrations (p<0.001). The NAC pretreatment significantly attenuated these alterations (p<0.001). Similarly, the administration of the *Tephrosia purpurea* extract improved all biochemical parameters in a dose-dependent manner. At 400 mg/kg, the extract significantly reduced the serum ALT, AST, ALP, and total bilirubin levels by 57.8%, 34.9%, 31.7%, and 82.0%, respectively, while it also significantly increased the albumin and total protein levels by 64.0% and 33.9%, respectively, compared with those of the CCl_4-treated (positive control) group (all p<0.001). The combination of NAC with the *Tephrosia purpurea* extract exerted the strongest effect, significantly reducing ALT, AST, ALP, and total bilirubin serum levels by 65.2%, 38.5%, 35.1%, and 87.6%, respectively, and significantly increasing the serum albumin and total protein levels by 81.5% and 40.7%, respectively, compared with those of the CCl_4-treated (positive control) group (all p<0.001). In conclusion, the *Tephrosia purpurea* extract can exert a significant, dose-dependent protection against CCl_4-induced hepatotoxicity, as evidenced by marked improvements in rat liver histology and hepatic function markers. The co-administration of this extract with NAC can further enhance these effects and provide a greater preservation of hepatic structure and function than either treatment alone.

Keywords

CCl_4; hepatoprotection; histopathology; NAC; *Tephrosia purpurea*

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PROCEEDINGS ABSTRACT

The impact of polymeric nanoparticle-based drug delivery systems in cancer therapy: a review

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Cancer remains one of the leading causes of morbidity and mortality, requiring the development of more effective and targeted therapeutic strategies. Given the fact that conventional chemotherapy is often associated with significant limitations (including poor selectivity, systemic toxicity, and nonoptimal pharmacokinetics), polymeric nanoparticle-based drug delivery systems are considered a promising approach to the enhancement of the efficacy and safety of anticancer therapies. These nanocarriers offer unique physicochemical properties that enable improved drug solubility, prolonged circulation time, and controlled release behaviour. This review aims to provide an up-to-date overview of the classification, targeting mechanisms, drug-loading approaches, advantages, limitations, and future potential of polymeric nanoparticle-based drug delivery systems in cancer therapy. Polymeric nanoparticles are broadly classified based on their structure, origin, and functionality. Structurally, they can be classified as nanospheres, nanocapsules, polymeric micelles, dendrimers, and nanogels. Nanospheres consist of a solid polymer matrix in which drugs are uniformly dispersed, whereas nanocapsules possess a core–shell structure that allows the encapsulation of therapeutic agents within a confined cavity. Polymeric micelles, formed from amphiphilic block copolymers, are particularly suitable for delivering hydrophobic anticancer drugs due to their core–shell architecture. Dendrimers, which are highly branched, tree-like macromolecules, provide multiple functional sites for drug attachment and surface functionalization; to this end, targeting ligands such as antibodies, peptides, folic acid, transferrin, aptamers, and small molecules are commonly used in order to enhance the selective binding to overexpressed receptors on cancer cells and to improve cellular uptake efficiency. Finally, nanogels are cross-linked hydrophilic polymer networks capable of high drug loading and responsive release. Based on origin, polymeric nanoparticles may derive from natural polymers such as chitosan and alginate, or synthetic polymers such as poly(lactic-co-glycolic acid) (PLGA), polyethylene glycol (PEG), and polylactic acid. Functionally, polymeric nanoparticles can be engineered in targeted, stimuli-responsive, or multifunctional systems. The mechanism of tumour targeting by polymeric nanoparticles involves passive and active processes. The passive targeting is primarily mediated through the enhanced permeability and retention effect, where nanoparticles preferentially accumulate in tumour tissues due to leaky vasculature and impaired lymphatic drainage. Alternatively, active targeting is achieved through ligand-mediated interactions with cancer cell receptors, followed by cellular uptake *via* endocytosis. The drug is then released intracellularly in response to tumour-specific stimuli such as an acidic pH, enzymatic activity, or redox conditions, thereby ensuring localized and controlled therapeutic action. Drug loading within polymeric nanoparticles can be accomplished through several strategies,

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including physical encapsulation within the polymer matrix, adsorption onto the nanoparticle's surface, or chemical conjugation to the polymer's backbone. Each method influences drug stability, release kinetics, as well as the overall therapeutic performance. Encapsulation is known to protect the drug from premature degradation, while conjugation enables more precise control over the release profiles. Advances in formulation techniques have enabled higher drug-loading efficiencies and have improved the nanoparticle systems' stability. Overall, polymeric nanoparticle-based drug delivery systems, especially those based on PLGA and PEG, represent a versatile and powerful approach for improving cancer therapy. However, certain challenges related to large-scale manufacturing, biological variability, immune recognition, and clinical translation remain obstacles to their clinical applicability.

Keywords

cancer therapy; drug delivery; PEG; PLGA; polymeric nanoparticles

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PROCEEDINGS ABSTRACT

Evaluating the barriers to glycaemic control in Iraqi patients with type 2 diabetes: the impact of lifestyle, medication adherence, and clinical inertia

Fatimah Abbas Hatif and Aymen A. Bash

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Achieving optimal glycaemic control is the cornerstone of effective type 2 diabetes mellitus (T2DM) management, as it is strictly required so as to prevent devastating micro- and macrovascular complications. Despite the availability of diverse therapeutic options, a substantial proportion of patients consistently fails to meet their clinical targets, thereby highlighting a significant gap in care. This cross-sectional study has aimed at determining the prevalence of uncontrolled T2DM among Iraqi patients and at systematically investigating the multi-level determinants contributing to treatment failure, by specifically focusing on lifestyle behaviours, prescribed drug regimens, and distinct barriers to medication adherence. The study took place between November 2025 and February 2026 across specialised diabetes management clinics, endocrinology consultation units, and primary healthcare centers located in the city of Hillah of the Babylon Governorate, in Iraq. Data were rigorously gathered through the use of a structured electronic questionnaire, supplemented by face-to-face interviews conducted during routine clinical visits in order to ensure data accuracy. Our study enrolled 158 adult patients, with an age range spanning from 20 to 60 years, comprising a demographic distribution of 51.9% women and 48.1% men. Strict inclusion criteria mandated that all participants possessed a confirmed, physician-documented T2DM diagnosis for a minimum duration of at least one year. The questionnaire of the study captured demographic data, anthropometric measurements, specific dietary habits, daily physical activity levels, as well as detailed medication-receiving histories. Based on the self-reported glycated haemoglobin (HbA1c) levels, participants were carefully stratified into two comparative cohorts: a "controlled T2DM" group (in which HbA1c levels were <7%) and an "uncontrolled T2DM" group (in which HbA1c levels were ≥7%). All collected data were subjected to rigorous statistical analysis utilizing the Chi-square test in order to determine statistical significance and to assess potential sex-based differences across all of the measured variables. Our analysis has revealed an alarmingly high prevalence of uncontrolled T2DM, negatively affecting 55.7% (N=88) of the total study population. Importantly, statistical evaluation confirmed no statistically significant variance regarding sex differences between the male and female cohorts in relation to their overall glycaemic control status. A detailed analysis of this uncontrolled patient cohort highlighted severe lifestyle-associated deficiencies; more specifically, 89.8% of these patients reported complete physical inactivity, 77.3% demonstrated persistent non-adherence to a low-carbohydrate dietary plan, while 54.5% were clinically classified as "obese". Moreover, the gathered clinical data exposed a substantial degree of clinical inertia that

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was firmly embedded within the local healthcare delivery framework. Despite failing to meet therapeutic glycaemic targets, 44.3% of the uncontrolled T2DM patients were inexplicably maintained on a single-agent (monotherapy) plan, and 44% remained on their currently prescribed drug regimen for over 18 consecutive months without receiving any necessary treatment intensification or dosage optimization. Notably, despite this observed clinical inertia, 64.7% of the uncontrolled T2DM patients expressed willingness to immediately initiate insulin therapy if explicitly recommended by their attending physician. Therapeutic non-compliance was conclusively identified in 35.3% of the poorly controlled T2DM group, driven primarily by unintentional factors like forgetfulness (41.9%), alongside an inability to tolerate bothersome medication side effects (25.8%). In conclusion, our study suggests that suboptimal glycaemic control is highly prevalent among Iraqi patients with T2DM, driven by a complex interplay of physical inactivity, obesity, widespread clinical inertia, and medication non-adherence, which necessitates immediate, collaborative multidisciplinary interventions involving physicians, clinical pharmacists, and dietitians in order to optimize treatment intensification and patient education.

Keywords

clinical inertia; HbA1c; Iraq; type 2 diabetes mellitus; uncontrolled diabetes

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PROCEEDINGS ABSTRACT

Pharmacodynamic study of several antibacterial–antifungal combinations against a *Staphylococcus aureus*–*Candida albicans* coinfection in an *in vitro* model: effects of albumin and platelets

Asmaa S. Dhaidan and Hussam W. Al-Humadi

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Optimizing antimicrobial treatment of bacterial–fungal coinfections remains a major clinical challenge due to the combined effects of the pathogen interaction and the host-related modifiers of drug activity. In particular, pharmacodynamic (PD) responses can be substantially altered by physiological factors such as protein binding and cellular components of the host environment, which are often not adequately captured in conventional *in vitro* models. This study was designed with the aim of quantitatively assessing how human albumin and platelets can influence the PD behaviour of combined antibacterial–antifungal therapies against *Staphylococcus aureus* and *Candida albicans*, using a dynamic *in vitro* pharmacokinetics / pharmacodynamics (PK/PD) model that simulates clinically relevant drug exposure. Such models have been shown to improve the translational prediction of antimicrobial efficacy [1]. Standard strains (*S. aureus* ATCC 25923 and *C. albicans* ATCC 90028), alone and in coinfection, were exposed to clinically relevant concentrations of antibacterial agents (vancomycin at 3 and 5 mg/L; clindamycin at 5 and 10 mg/L) administered as combination therapies with antifungal agents (caspofungin at 10 and 20 mg/L; amphotericin B at 2.5 and 5 mg/L). Experiments were conducted in the absence and presence of 2% human albumin and / or 2% platelet-rich plasma so as to mimic physiological conditions. Subsequently, the antimicrobial activity was assessed through relative optical density measurements and expressed in growth inhibition scales, with PD parameters estimated using a sigmoidal maximum effects (E_{max}) model and the area under the curve (AUC). The exposure–response relationships were defined using the $fAUC_{0–24}/C_{max}$ index, demonstrating an excellent model fitting (mean R^2 being ≈0.9) that is consistent with those of PK/PD analyses of antimicrobial combinations in previous studies [2]. Across all experimental conditions, combination therapies exhibited a concentration-dependent antimicrobial activity with characteristic sigmoidal E_{max} profiles. Notably, quantitative analyses revealed consistent and significantly augmented E_{max} values in the presence of host factors. Platelets markedly augmented the antimicrobial activity across all regimens, which may be attributed to the platelets essentially acting as key innate immune cells that upon activation by microorganisms or inflammatory mediators, release antimicrobial peptides from their granules in order to kill pathogens. This effect was further influenced by albumin, while the high-

Dhaidan A. S., Al-Humadi H. W.: Pharmacodynamic study of several antibacterial–antifungal combinations against a *Staphylococcus aureus*–*Candida albicans* coinfection in an *in vitro* model: effects of albumin and platelets. *Colloq. Erud.* 1: 89–90 (2026).
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est E_{max} values were consistently observed in conditions combining an exposure to both albumin and platelets (p<0.001), thereby supporting previous findings on the host-mediated modulation of antimicrobial activity [3]. Among the tested regimens, vancomycin plus caspofungin demonstrated the highest intrinsic antimicrobial activity across mono- and coinfection models, achieving maximal E_{max} values. In contrast, vancomycin plus amphotericin B showed comparatively lower maximal activity, but maintained more stable PD responses across the assessed *in vitro* host conditions. On the other hand, the clindamycin-based combinations exhibited intermediate antimicrobial effects, but were significantly influenced by the host-related factors. Finally, in the coinfection models, all combinations exhibited reduced PD activity when compared to single-organism conditions, confirming the negative impact of polymicrobial interactions on treatment efficacy. Overall, this study suggests that while combination antimicrobial therapies can enhance the antimicrobial activity against bacterial–fungal coinfections, host-related factors (namely, protein binding and platelet interactions) can substantially influence the antimicrobial PD outcomes. These findings also underscore the importance of incorporating physiological conditions into the *in vitro* PK/PD models so as to improve the translational relevance of the generated data and to optimize therapeutic strategies for polymicrobial infections.

Keywords

albumin; antibacterial–antifungal synergy; pharmacodynamic model; platelets; polymicrobial infection

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PROCEEDINGS ABSTRACT

Suicidal ideation as an adverse effect of fluoroquinolones: a review of recent evidence

Zahraa Hazim, Apostolos Zarros, Rafal J. Al-Saigh, Manal M. Younus, and Hussam W. Al-Humadi

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Fluoroquinolones (including widely used agents such as ciprofloxacin, delafloxacin, levofloxacin, moxifloxacin, and ofloxacin) represent an important tool of modern therapeutics. Due to their tissue penetration ability, favourable pharmacokinetic profiles, and bactericidal activities against a broad spectrum of Gram-positive and -negative pathogens, these antibiotics remain clinically important for selected serious infections. However, the historical perception of fluoroquinolones as having benign safety profiles is now being challenged by a growing body of clinical evidence. Over the last decade, this shifting landscape has redirected medical focus toward a spectrum of severe (and often debilitating) neuropsychiatric adverse drug reactions, with global regulatory authorities (including the FDA) issuing warnings in order to highlight the potential of this class of antibiotics for causing serious central nervous system (CNS) toxicities. Suicidal ideation and suicidal behaviour have been recognised by regulatory product information as rare potential outcomes of fluroquinolone-associated psychiatric reactions, although neither their frequency nor their period of risk can currently be quantified. This narrative review attempts to synthesize current clinical evidence, pharmacovigilance data, and the findings of mechanistic research in order to assess the intricate relationship between fluoroquinolones and the risk of severe neuropsychiatric adverse events [1]. We have, herein, placed particular emphasis on assessing the fluoroquinolone-associated risks of suicidal ideation as well as those of acute psychosis and major depression. Clinical evidence reveals a distressing pattern of rapid-onset psychiatric decompensation, and several cases indicate that severe neuropsychiatric symptoms can manifest even after a few doses of fluoroquinolones in patients with no prior history of psychiatric disease or identified psychological vulnerabilities. Case reports and some spontaneous-reporting analyses support a potential association between fluroquinolones and serious psychiatric reactions, including suicidal ideation; however, the findings from pharmacovigilance and population-based studies are inconsistent, and the incidence and magnitude of any excess risk remain uncertain [2,3]. The reported symptoms exist on a broad and unpredictable continuum that ranges from insidious insomnia and mild anxiety to profound psychomotor agitation, vivid hallucinations, and suicidal behavior. Potential susceptibility factors described in the literature include advanced age, renal impairment, concomitant medicines, and pre-existing neuropsychiatric conditions; however, evidence identifying reliable predictors of suicidal ideation remains limited. Proposed mechanisms include antagonism of gamma-aminobutyric acid-mediated inhibitory neurotransmission, altered excitatory neurotransmission involving *N*-methyl-D-aspartate receptors, and oxidative or mitochondrial effects. However, the relative contribution of these mechanisms to individual psychiatric reactions, particularly suicidal ideation, remains uncer-

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tain. Until future studies manage to identify genetic markers and / or biological signatures that can accurately predict individual susceptibility to these rare but life-threatening fluoroquinolone-induced neuropsychiatric adverse events, clinicians will need to prioritize the identification of high-risk patient phenotypes and to exercise judicious prescribing practices for this class of antibiotics, particularly in the cases of uncomplicated infections for which alternative therapeutic options exist. Patients and caregivers should also be counselled to seek immediate medical advice if new or worsening psychiatric symptoms (including depression, psychosis, or suicidal thoughts) occur, and treatment should be discontinued promptly when a serious psychiatric reaction develops.

Keywords

adverse drug reaction; fluoroquinolones; neuropsychiatric symptoms; pharmacovigilance; suicidal ideation

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