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ЗАПОРІЗЬКИЙ ДЕРЖАВНИЙ МЕДИКО-ФАРМАЦЕВТИЧНИЙ
УНІВЕРСИТЕТ**

**КООРДИНАЦІЙНА РАДА З НАУКОВОЇ РОБОТИ СТУДЕНТІВ, АСПІРАНТІВ,
ДОКТОРАНТІВ І МОЛОДИХ ВЧЕНИХ
СТУДЕНТСЬКА РАДА**

ЗБІРНИК ТЕЗ ДОПОВІДЕЙ

**86 ВСЕУКРАЇНСЬКОЇ НАУКОВО-ПРАКТИЧНОЇ КОНФЕРЕНЦІЇ
МОЛОДИХ ВЧЕНИХ ТА СТУДЕНТІВ З МІЖНАРОДНОЮ УЧАСТЮ**

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ДЕЯКІ МЕХАНІЗМИ КАРДІОПРОТЕКТИВНОЇ ДІЇ ФІКСОВАНОЇ КОМБІНАЦІЇ ТІОТРИАЗОЛІНУ ТА АРГІНІНУ НА МОДЕЛІ ЕКСПЕРИМЕНТАЛЬНОЇ ХРОНІЧНОЇ АЛКОГОЛЬНОЇ ІНТОКСИКАЦІЇ	287
<i>Терещенко М.С.</i>	
НЕЙРОПРОТЕКЦІЯ ПРИ ЕКСПЕРИМЕНТАЛЬНОЇ ЧЕРЕПНО-МОЗКОВІЙ ТРАВМІ (ЧМТ), ПОСДНАНІЙ З АКУБАРОТРАВМОЮ	288
<i>Сахно О.С., Печорська М.М.</i>	
SOME ASPECTS OF THE APPROACH TO PAIN MANAGEMENT IN PAEDIATRIC DENTAL PRACTICE	289
<i>Filchak A.O.</i>	
ПЕРОРАЛЬНІ ТА ІН'ЄКЦІЙНІ СТРАТЕГІЇ КОРЕКЦІЇ ІНСУЛІНОРЕЗИСТЕНТНОСТІ.....	290
<i>Гоблик Н.М.</i>	
ВИВЧЕННЯ РІВНЯ ОБІЗНАНОСТІ ЩОДО ПРОГРАМИ «ДОСТУПНІ ЛІКИ» СЕРЕД ЦІЛЬОВОЇ АУДИТОРІЇ	291
<i>Баранова К.О.</i>	
ФІЗІОЛОГІЧНІ ТА БІОХІМІЧНІ ПРОЦЕСИ У ПОРОЖНИНІ РОТА ЯК ФАКТОР ПІДТРИМАННЯ ГОМЕОСТАЗУ ТВЕРДИХ ТКАНИН ЗУБА	293
<i>Данилюк О.С.</i>	
СЕРОТОНІНОВИЙ СИНДРОМ	293
<i>Демко А.О., Голавська Д.В.</i>	
НОВІ ПІДХОДИ ДО АНТИКОАГУЛЯНТНОЇ ТЕРАПІЇ ПРИ ФІБРИЛЯЦІЇ ПЕРЕДСЕРДЬ: ПОРІВНЯЛЬНИЙ АНАЛІЗ ПРЯМИХ ПЕРОРАЛЬНИХ АНТИКОАГУЛЯНТІВ ТА ВАРФАРИНУ	294
<i>Мельник Д.О.</i>	
ЕКСПЕРИМЕНТАЛЬНЕ ДОСЛІДЖЕННЯ ЕФЕКТИВНОСТІ АІ-АСИСТЕНТА У СТВОРЕННІ ТЕСТІВ З ФАРМАКОЛОГІЇ	295
<i>Михалюк С.Л.</i>	
ЕФЕКТИВНІСТЬ АВТОМАТИЗАЦІЇ ЗНАНЬ У ФАРМАКОЛОГІЇ: ЗАСТОСУВАННЯ ШТУЧНОГО ІНТЕЛЕКТУ ТА ФЛЕШ-КАРТOK.....	296
<i>Присяжнюк А.О.</i>	
ЧИ ЕФЕКТИВНИЙ АІ-ЧАТ-БОТ У ВИВЧЕННІ ФАРМАКОЛОГІЇ? ЕКСПЕРИМЕНТАЛЬНИЙ ПІДХІД..	297
<i>Сушко А.С.</i>	
БЛОКАДА РААС ЯК ФУНДАМЕНТ ТЕРАПІЇ СЕРЦЕВОЇ НЕДОСТАТНОСТІ: РОЛЬ ІНГІБІТОРІВ АПФ У РЕМОДЕЛЮВАННІ МІОКАРДА	298
<i>Пацьо В.В.</i>	
НАНОФАРМАКОЛОГІЧНІ ПІДХОДИ ДО ДОСТАВКИ ПРОТИПУХЛИННИХ ПРЕПАРАТІВ У ЦНС НА ПРИКЛАДІ ЛІПОСОМАЛЬНОГО ДОКСОРУБІЦИНУ	299
<i>Добра К.В.</i>	
CHELIDONIUM MAJUS ALKALOIDS IN CANCER PREVENTION AND TREATMENT: CURRENT STATUS AND FUTURE PROSPECTS.....	300
<i>Levenko M.O.</i>	
PHARMACOLOGICAL CORRECTION OF THE NITRIC OXIDE SYSTEM AND NITROSATIVE STRESS IN MYOCARDIUM FOLLOWING PRENATAL HYPOXIA	301
<i>Popazova O.O., Makarovych V.O.</i>	
CLINICALLY RELEVANT DRUG-DRUG INTERACTIONS IN THE TREATMENT OF HIV INFECTION IN PATIENTS WITH CONCOMITANT TUBERCULOSIS	302
<i>Yefremova V.V.</i>	
CHANGES IN LABORATORY AND FUNCTIONAL PARAMETERS IN LABORATORY ANIMALS DURING SEPSIS MODELING AND EXPERIMENTAL THERAPY	303
<i>Birch, A., Shybka, S., Udovychenko, V.</i>	
ПОТЕНЦІЮВАННЯ ПРОТИЗАПАЛЬНОЇ АКТИВНОСТІ НЕСТЕРОЇДНИХ ПРОТИЗАПАЛЬНИХ ПРЕПАРАТІВ ЗА УМОВ ЕКСПЕРИМЕНТАЛЬНОГО АД'ЮВАНТНОГО АРТРИТУ ШЛЯХОМ КОМБІНОВАНОГО ЗАСТОСУВАННЯ ІЗ БЕЗКЛІТИННИМИ КРІОКОНСЕРВОВАНИМИ БІОЛОГІЧНИМИ ЗАСОБАМИ	304
<i>Студент В.О., Гладких Ф.В., Матвєєнко М.С.</i>	

CHELIDONIUM MAJUS ALKALOIDS IN CANCER PREVENTION AND TREATMENT: CURRENT STATUS AND FUTURE PROSPECTS

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Background: Plant-derived alkaloids products are of crucial importance in medicine especially in the cancer arena. Plant alkaloids used as antitumor drugs are primarily divided into vinca alkaloids, taxanes, camptothecins, and podophyllotoxin derivatives. They act mainly by disrupting cell division (*microtubule inhibition*) or DNA replication. Key examples include *Vincristine*, *Vinblastine*, *Paclitaxel*, *Irinotecan* and *Etoposide*. The greater celandine (*Chelidonium majus*) contains isoquinoline alkaloids, primarily *Chelidonine*, *Tetrandrine*, *Sanguinarine*, and *Chelerythrine* that show significant anticancer potential by inducing apoptosis (*programmed cell death*) and inhibiting tumor growth in various cancers, including pancreatic, colon, liver, and melanoma.

Objective: To evaluate current data from scientific sources to determine the molecular mechanisms, therapeutic potential, and sex-specific differences associated with *Chelidonium majus* alkaloids in cancer prevention and treatment.

Materials and Methods: A literature review was conducted using peer-reviewed articles, cohort studies (EPIC-Heidelberg, Japan Collaborative Study), and clinical data sourced from PubMed, ScienceDirect, MDPI, Google Scholar and SpringerLink, and other scientific databases.

Results: The greater celandine (*Chelidonium majus*) has long been recognized in traditional medicine for its therapeutic potential, and modern research is increasingly validating its anticancer properties. Greater celandine contains over 70 pharmacologically active isoquinoline alkaloids (0.27–4 % concentration), with major antitumour compounds including *Chelidonine*, *Sanguinarine*, and *Chelerythrine*. These compounds induce apoptosis (*programmed death*) of cancer cells and inhibiting their division, growth, and metastasis, and overcome multi-drug resistance by targeting mitochondrial death pathways and inhibiting the expression of multidrug resistance gene 1 (*MDR-1*, or *P-glycoprotein*), often acting on cancer cells with moderate selectivity.

The Celandine Alkali Injection Formula (CAIF) offers benefits on clinical internal medicine treatments, within which *Chelidonine*, *Tetrandrine*, *Sanguinarine*, and *Chelerythrine* are recognized as potential quality markers. Chelidonine, a major isoquinoline alkaloid from *Chelidonium majus*, demonstrated anticancer effects through multiple pathways: inducing apoptosis via mitochondrial disruption and caspase activation, generating reactive oxygen species (ROS), inhibiting angiogenesis by downregulating vascular endothelial growth factor (VEGF), and modulating immune responses by enhancing macrophage and dendritic cell activity. Chelidonine elevates ROS levels, causing oxidative DNA damage and prompting mitochondrial-mediated apoptosis in cancer cells.

Tetrandrine, a bisbenzylisoquinoline alkaloid, act as a potent anti-inflammatory and anti-tumor agent by inhibiting pro-inflammatory cytokines such as IL-6 and TNF- α . It suppressed cancer metastasis by inhibiting IL-6-induced cell migration, invasion, and epithelial-mesenchymal transition in various cancers. Tetrandrine demonstrated anticancer activities in patients with lung cancer, colon cancer, gastric cancer, breast cancer, bladder cancer, prostate cancer, ovarian cancer, pancreatic cancer, cervical cancer and liver cancer. The molecular mechanisms of cytotoxic action of tetrandrine are related to its properties to induce cancer cell apoptosis, autophagy and cell cycle arrest: inhibit cell proliferation, migration and invasion, ameliorate metastasis and suppress tumour cell growth. Pharmaceutical applications of tetrandrine combined with nanoparticle delivery system including liposomes, microspheres and nanoparticles with better therapeutic efficiency have been designed and applied encapsulate tetrandrine to enhance its stability and efficacy in cancer treatment.

Chelidonium majus alkaloids can enhance the antitumor activity of antitumour agents such as vinblastine, particularly when looking at delayed treatment outcomes in lymphoma. Preclinical studies indicate that combining celandine alkaloids with traditional chemotherapy (e.g., *Colchicine*, *5-Fluorouracil*, *Vincristine* and *Vinblastine*) improves tumor suppression, enhances apoptosis through the mitochondrial pathway, and helps reduce the growth of resistant tumor cells. These alkaloids, particularly, induce cancer cell death by upregulating tumor suppressor genes, including p53 and p21.

Sanguinarine and Chelerythrine demonstrated strong cytotoxic activities against various cancer cell lines, including PANC02 (*pancreatic*) and HeLa (*cervical*).

Conclusions: Greater celandine (*Chelidonium majus*) isoquinoline alkaloids *Chelidonine*, *Tetrandrine*, *Sanguinarine*, and *Chelerythrine* demonstrated anticancer effects through multiple pathways: apoptosis activation via mitochondrial disruption and caspase induction, generating ROS, inhibiting angiogenesis by downregulating VEGF, and modulating immune responses by enhancing macrophage and dendritic cell activity. Greater celandine alkaloids, especially chelidonine and tetrandrine, are substantial potential as adjunctive anticancer agents. Their multi-target effects, including enhancing apoptosis, reducing angiogenesis, limiting tumor blood supply, and inhibiting tumor cell proliferation, are enhanced when combined with standard treatment methods.

PHARMACOLOGICAL CORRECTION OF THE NITRIC OXIDE SYSTEM AND NITROSATIVE STRESS IN MYOCARDIUM FOLLOWING PRENATAL HYPOXIA

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Purpose: The study aimed to investigate the influence of specific pharmacological agents on the nitric oxide system and the intensity of nitrosative stress within the myocardium of rats subjected to prenatal hypoxia (PH). Key parameters included the expression of endothelial (eNOS) and inducible (iNOS) NO synthase isoforms, alongside stable NO metabolite levels in 1- and 2-month-old animals.

Materials and methods. The experiments were conducted on Wistar rats. Intrauterine hypoxia was induced by intraperitoneal administration of sodium nitrite (50 mg/kg) to pregnant dams during the final trimester. This model replicates clinical intrauterine hypoxia by compromising placental gas exchange rather than through direct systemic toxicity. Postnatal offspring were categorized into six cohorts: an intact group, a PH-control (untreated), and four groups receiving daily intraocular administration of thiotriazoline, angioline, L-arginine, or mildronate for 30 days. Biomaterial was harvested on the 30th and 60th days of development. Analytical methods included enzyme-linked immunosorbent assay (ELISA) for protein expression and the Griess method for quantifying NO metabolites.

Results. Findings indicate that PH triggers enduring nitroxidergic dysfunction in the rat heart across both observed timeframes. Observed pathologies included a depletion of stable NO metabolites, suppressed eNOS levels, and upregulated iNOS expression. Coupled with elevated nitrotyrosine, these markers signify reduced nitric oxide bioavailability and its diversion toward toxic peroxynitrite formation. While all tested modulators exhibited protective effects, angioline demonstrated superior efficacy. It facilitated a nearly threefold increase in eNOS expression while halving iNOS levels. Furthermore, angioline reduced myocardial nitrotyrosine by 60% and boosted stable NO metabolites by 81%.

Conclusions. Angioline enhances NO bioavailability by promoting its stabilization through S-nitrosothiol complexes with glutathione and cysteine. Thiotriazoline also showed significant potency in re-establishing the eNOS/iNOS equilibrium. In contrast, L-arginine provided only moderate benefits, while mildronate proved the least effective, failing to influence eNOS and leaving signs of residual nitrosative stress and regulatory impairment.