junior college (Junior Colleges). When entering a pharmaceutical university, it is mandatory to pass the Pharmacy College Admission Test, which is divided into 6 sections: verbal abilities, knowledge in chemistry and biology, computational skills, reading comprehension and two written sections.

Japanese pharmaceutical education devotes a significant part of educational time to subjects related to the methodology of drug development, such as bioanalytical chemistry, mechanism of action of drugs, pharmacokinetics, pharmaceutical chemistry, toxicology, development of new drugs, targeted synthesis. Minimal attention is paid to the humanities.

Therefore, we can make a conclusion that obtaining such information will help to borrow the experience of world universities, as well as to create own methods of education that will promote faster development of the pharmaceutical industry through the education of highly qualified pharmacists in Ukraine.

Shchudrova T.S.

THERAPEUTIC POTENTIAL AND PERSPECTIVE ON MELATONIN USE FOR DRUG-INDUCED NEPHROPATHY

Department of Pharmacology
Bukovinian State Medical University

The research aimed to review the effects and mechanisms of action of the pineal hormone melatonin, to study the nephroprotective effect of exogenous melatonin in conditions of drug-induced nephropathy, and to assess the prospects of its use for prevention and treatment of kidney diseases based on the literature data and results of own studies. According to literature and results of our previous research, the nephroprotective effect of melatonin has been shown in various experimental models of acute renal injury (AKI). Numerous studies established the antioxidant, anti-inflammatory, anti-apoptotic, immunomodulatory, and cytoprotective effects of melatonin, and showed its ability to restore the function and structure of the kidneys.

The objective of the current study was to evaluate the effects of melatonin (5 mg/kg) on the animal model of acetaminophen-induced AKI. The experiments were conducted on nonlinear mature white rats weighing 150-200 g, and randomly distributed into three groups (n=7). Group I – control; group II – acetaminophen-induced AKI (administration of paracetamol (Health, Ukraine) at a dose of 750 mg/kg); group III – administration of melatonin (Sigma-Aldrich, USA) at a dose of 5 mg/kg against the background of AKI development. Animals were withdrawn from the experiment 24 h later, while blood, urine and kidneys were sampled for biochemical and histopathological assessments. Statistical processing of the obtained data was performed using the SPSS Statistics 17.0 software.

In our experiment, a single administration of the toxic acetaminophen dose to rats (group II) resulted in drug excessive accumulation and damage to the proximal tubular cells. It is known, that cellular toxicity of acetaminophen is associated with translocation and dysfunction of Na+-K+-ATPase, which ensures effective sodium reabsorption. In rats with acetaminophen-induced AKI a decrease in sodium reabsorption and, accordingly, an increase in fractional sodium excretion was found. An increase in the sodium concentration in the tubular fluid led to the activation of tubuloglomerular feedback with a 2-fold decrease in glomerular filtration rate (GFR), reduced urine output, and development of retention azotemia. Significant proteinuria compared to the control confirms the severe toxic damage to renal tubular cells. In animals that received melatonin, treatment (group III) renal dysfunction was less pronounced. Melatonin counteracted the nephrotoxic effect of acetaminophen, as evidenced by the prevention of significant sodium loss due to maintenance of the reabsorption capacity of tubular cells, restoration of urine output due to maintenance of GFR, and prevention of retention azotemia and significant proteinuria. Acetaminophen overdose induced the oxidative stress from the intensification of ROS production, lipid and protein peroxidation processes and the simultaneous decline of the enzymatic antioxidant capacity. In animals from group II, a significant increase in the level of lipid peroxidation end-product malondialdehyde (MDA) and protein oxidative modification products (OMP) was found in kidney tissue ($p<0.05$ compared to the control group). Acetaminophen also compromised local

antioxidant system, manifested in a decrease in glutathione peroxidase (GPx) and catalase (CAT) activity ($p<0.05$ compared to the control group). Melatonin showed a significant antioxidant effect manifested in attenuation of both lipid and protein peroxidation in the kidney tissue, along with an increase in the GPx and CAT activity compared to untreated animals ($p<0.05$).

The obtained results show the ability of melatonin to reduce the severity of damage and prevent kidney dysfunction associated with acetaminophen over dose. Treatment with melatonin was suppressed the progression of oxidative stress in kidney tissue through the limitation of lipid and protein peroxidation and activation of the key antioxidant enzymes. Results of research complement to existing data on the nephroprotective activity of melatonin and substantiate the high therapeutic potential and prospects of melatonin use as adjunctive therapy of drug-induced nephropathy.

Shliusar O.E.

VOLTAMPEROMETRIC DETERMINATION OF THIORIDAZINE AS ITS S, S'-DIOXIDE, OBTAINED BY CARO ACID

Department of Pharmacy
Bukovynian State Medical University

Known synthetic drug thioridazine (syn. thioridazine hydrochloride, ridazine, sonapax, melleryl, tioryl) belongs to the original piperidine fentanyl and is widely used as a neuroleptic, sedative, thymoleptic and sedative drug in medical practice. It detects a mild antidepressant effect. The most effective disorders are accompanied by fear, stress, and excitement. The dose is 50-100 mg per day. Medicine is produced in tablets of 10, 25 and 100 mg for children - 0.2% suspension and syrup. For determination of basic substance content in the substance acidimetric method in the medium of glacial acetic acid and acetic anhydride (potentiometric titration) is recommended, in tablets and pills – direct UV spectrophotometry method.

The aim of our study was to develop a simple, selective and fast enough, and cost-effective way to assay thioridazine at 10 mg tablet sonapax, produced at pharmaceutical plant AT (Jelenia Góra, Poland), based on previous drug oxidation in an acidic medium using potassium hydrogenperoxomonosulfate to the corresponding S, S'-dioxide with the subsequent voltammetric determination of its recovery after wave of mercury drops at -0,41 B (SCE). Formation in the studied reactions S,S'-dioxide is due to electrophilic attack β-oxygen atom of peroxoacid peroxide group on sulfur atoms of during minute. In the process of electrochemical reduction polarograms S,S'-dioxide thioridazine experienced two waves of E_n: at - 0.41 V (restoration to the S-oxide) and slightly less at - 0.72 V (SCE), height is proportionally increased depending on the concentration of the analyte. As we have chosen the analytical wave with peak potential at - 0.41 V (SCE). It was experimentally found that the dependence of peak current strength of the recovery potentials S,S'-dioxide thioridazine in - 0.41 V (I, mA) on the concentration (C, mol / l) in the concentration range from $2.0 \cdot 10^{-5}$ to $1{,}6 \cdot 10^{-4}$ mol / l by the equation: $I = (0{,}18 \pm 0{,}03) \cdot 10^{5} \cdot c$ (correlation coefficient r = 0,98). The content of thioridazine by a method of the standard was determined. The reproduction of the signal (peak height of the current restoration of thioridazine potential - 0.41 V (SCE) in the test solution Rcc thioridazine hydrochloride $7{,}37 \cdot 10^{-5}$ mol / l (10.00 ml of the drug taken for analysis) characterized by the RSD = 3,27 for $n = 5$; $P = 0{,}95$).

Therefore, the method for quantitative determination of thioridazine tablets of 0.01 g by variable-current voltammetry method as S, S'-dioxide thioridazine (E_n = - 0.41 V (SCE) obtained by using Caro acid is developed. RSD = 3,27% ($n = 5$, $P = 0{,}95$). The results were in good agreement with those of BhP (δ = -1,01%).