## СЕКЦІЯ 4
## АКТУАЛЬНІ ПИТАННЯ ХРОНОБІОЛОГІЇ ТА ХРОНОМЕДИЦИНИ

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### CHANGES OF MORPHOFUNCTIONAL STATE OF MEDIAL SMALL-CELL SUBNUCLEUS OF PARAVENTRICULAR HYPOTHALAMIC NUCLEUS ON THE BACKGROUND OF DIFFERENT ILLUMINATION PERIODS

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Nowadays the study of implication of neuroendocrine structures for central mechanisms of circadian rhythms is one of the most actual questions of modern chronophysiology. The changes of photoperiod, being a stress factor, desynchronize the rhythmicity of somatic and visceral function, coordination and modulation of body adaptation to various influences. Medial small-cell subnucleus of paraventricular hypothalamic nucleus (msPVN) is one of the structures, that are primary involved in neuroendocrine response in case of stress reactivity, regulating the activity of adenohypophysis by synthesis of corticotrophin-releasing hormone. The data, concerning morphofunctional characteristics of msPVN, exposed to photoperiod of different duration, hasn't been reported in literature.

The aim of the research was to reveal the influence of steady lighting on morphofunctional state of msPVN in different day intervals. Sexually mature rat males were divided for two groups: first group was subjected to standard lighting (light input from 8.00 to 20.00), second one – to 7-days lighting. Morphometry and densitometry of msPVN, quantitation of their RNA content were conducted by computerized image analyzer VIDAS-386 (Germany) within visible spectrum. Measured at 14.00 and 02.00, msPVN indices of rats, kept in hyperilluminized cages, weren't affected. The exception concerned RNA concentration in nucleolus, that was by 2,5% higher in the daytime and by 2,7% lower at night as compared with controls. The analysis of daily variations and rhythmicity of msPVN neurons functional activity in rats under photostimulation revealed them to be similar to those of intact animals. Steady lighting at 14.00 led to the increase of neuron area by 7,8% related to augmented area of nucleus and cytoplasm (by 7,4 and 16,2% respectively) in comparison with the group of previous time interval.

Though long-time lighting is thought to be stress factor and trigger of desynchronism, it doesn't concern the studied structures. Absence of accelerated msPVN functional activity and significant differences in neuron area under steady and standard lighting on $8^{th}$ day indicates the implication of adaptive-compensatory mechanisms, directed to maintain the stability of msPVN, and the impossibility of alteration of their regulation under light irritant.

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### CURRENT ISSUES IN THE STUDY OF PRION INFECTIONS

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The prion proteins were discovered in 1982 and 2021 marks the 39th anniversary of the first findings in etiology of especially dangerous "slow" infections – prion diseases in humans and animals. The establishment of the role of infectious prion protein (PrPd) as the only factor in the pathogenicity of prion diseases turned out to be shocking to the scientific community, but subsequently changed the approach to research even in such conformational diseases as Alzheimer's disease, Parkinson's disease, diabetes mellitus and others, the pathogenesis of which is characterized by pronounced amyloid formation – protein aggregates including densely packed β-layers. Taking into account that over the past years considerable knowledge has been accumulated about the biology of prion proteins and the pathogenesis of the diseases they cause, the aim of this work is to characterize modern issues of prion infections for human and animals.

The formation and accumulation of the cellular prion protein PrPc is the result of the expression of the PRNP gene localised in human chromosome 20. In various animal species, the

length of its polypeptide chain varies slightly: from 252 amino acid residues in a rabbit to 253-254 in human. The presence of a 22-membered signal sequence at the N-terminal region of the PrPc protein molecule provides cotranslational transfer of the newly formed polypeptide chain across the membrane of the endoplasmic reticulum. When the polypeptide chain passes through the channel (SEC61) in the rough ER membrane wall, the leader signal sequence is removed, as well as the folding of the molecule into a globule and its covalent modification. Two N-glycosylation sites of the sequense Asn-Ile-Thr and Asn-Phe-Thr are located at amino acid residues 181 and 197 respectively (human prion protein). The distribution of di-, mono-, and non-glycosylated forms of prion can vary both in different organisms of the same species, and within the same organism. Glycosylated forms of the protein are also diverse, which makes it possible to isolate about 400 different PrPc glycoforms. Natural and induced mutations in the hydrophobic sequence located in the middle part of the molecule, near it, as well as in the region of the N-terminal region, lead to an increase in the proportion of CtmPrP, which causes neurodegenerative diseases. A number of facts have been established that indicate the influence of the degree of glycosylation of prion proteins on the efficiency of prion disease transmission, as well as on the formation process of various strains of the prion pathogen. To date, more than 30 possible mutations of the prion protein have been described, of which most are reliably associated with hereditary prion diseases in human. Together with the infectious isoform, they do not exceed 10-20% of all reported cases of prion diseases, the rest are the sporadic form of Creutzfeldt-Jakob disease (neurodegenerative disease of spongiform encephalopathy in humans).

Thus, prion diseases are a group of neurodegenerative diseases of animals and humans caused by infectious isoforms of one of the host proteins called prion (PrP) and encoded by the cellular genome. Currently, the term PrP is used both to denote the isoform of a protein formed during normal cell metabolism (PrPc) and its pathological (infectious) isoform (PrPd), which causes prion diseases in humans and various animal species.

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## NEW APPROACHES IN DIAGNOSIS OF CYSTIC FIBROSIS

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Cystic fibrosis (CF) is an autosomal recessive disorder caused by the mutation of a gene located on the long arm of chromosome 7 that encodes for a protein of 1480 amino acids, the cystic fibrosis transmembrane conductance regulator (CFTR), which works as a chloride channel on the apical membrane of epithelial cells. This mutation results in a change of the viscosity of secretions, and the production of thick mucus that leads to malabsorption, loss of electrolytes in sweat, and alteration of pulmonary secretions. As we know along with impairment of sweat glands and mucous glands the patient also suffers from various allergic bronchopulmonary infections such as aspergillosis and are at a high risk of pneumothorax.

Purpose of the study - to find out the most prevalent methods used to diagnose Cystic fibrosis by deep surfing scientific internet sources. To explain the main diagnostic that are used for Cystic fibrosis along with their importance, methods, and comparison between their cost and effectiveness.

Immunoreactive trypsinogen (IRT) test is the Newborn Screening technique used to diagnose cystic fibrosis in newborns. This test is done by taking blood sample by pricking the heel of the infant. If the level of IRT is not abnormal, then it is possible that the newborn is not suffering from CF. But, if the infant shows signs and symptoms consistent with CF, other tests for cystic fibrosis, such as sweat chloride or CF gene mutation testing, can be considered. Such as Sweat chloride test. It is well known that level of chloride in sweat is high in patients with CF. It happens due to defective chloride transport. The sweat test detects the level of chloride that is excreted in sweat. It is used as a diagnostic technique for CF. A chloride level of more or equal to 60 mmol/L is likely to be diagnosed with CF. Next test that is commonly used for CF is a Sputum test. Patients with CF frequently suffers from respiratory infections, caused by bacteria or fungi. A