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Матеріали 101 – ї підсумкової наукової конференції професорсько-викладацького персоналу вищого державного навчального закладу України «Буковинський державний медичний університет» (м. Чернівці, 10, 12, 17 лютого 2020 р.) – Чернівці: Медуніверситет, 2020. – 488 с. іл.

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У збірнику представлені матеріали 101 – ї підсумкової наукової конференції професорсько-викладацького персоналу вищого державного навчального закладу України «Буковинський державний медичний університет» (м.Чернівці, 10, 12, 17 лютого 2020 р.) із стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

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IMPACT OF SEASONS ON CIRCADIAN RHYTHM

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According to the recent researches, light is the most potent time cue for phase-shifting circadian rhythms, but the period and amount of solar irradiation vary dynamically with season, especially in regions far from the equator. There is evidence that chronotype is modified by seasonal change, most likely due to the changes in the light environment, but interindividual differences in photoperiod responsiveness mean that some people are more affected than others. A study of circadian rhythm influence on the human heart rate, depending on a season changes, was the aim of our research.

The study was conducted on 20 students during summer-winter seasons to observe impact of season changes on biological clock mechanism by recording the waking, sleeping time and heartbeat rate. The most obvious and reliable feature of seasons is the change in daylength or photoperiod with shorter photoperiods during winter months and longer photoperiods during summer (Shawa N., Rae D.E., Roden L.C., 2018). It was found that sleep duration was longer in winter than in summer photoperiods and that the longer sleep was associated with longer duration of melatonin secretion (Wehr T.A., 1991). Secretion of melatonin reaches its peak at the middle of the night and decreases throughout the day, its presence provides information about night-length. HRV increased during the night in particular and a nighttime peak during the second half of the night was identified (Sammito S., Sammito W., Böckelmann I., 2016). During autumn the time of waking was almost the same. Here waking means that it can be a remainder that the person woke the same time on the previous day. Heart beating when persons woke was comparatively higher than when they slept: it is around 106-116 times before sleep and 126-135 times during waking up. The duration of sleep was in the range of 6-8 hours.

The results of conducted experiments are different in winter as well as in a dark room indicating the dependence of biological clock activity in response to external stimuli, namely solar light at different seasons. Persons during winter take a little more time to wake up (10 hrs). In addition, heart works harder to keep body warm due to a cold weather. Thus, it is clear that biological clock depends on the season changes and plays a major role in the regulation of human heart health.

Lomakina Yu.V.

CHARACTERISTIC OF PRO-INFLAMMATORY AND ANTI-INFLAMMATORY CYTOKINES IN KIDNEY FAILURE

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It is known that immune system is activated by a diversity of factors, including pathogens, damaged cells and toxic compounds. These factors may induce acute and/or chronic inflammatory responses in the kidney, liver, heart, pancreas, lung, brain, intestinal tract and reproductive system, altogether causing injure of tissue or disease. The etiologies of inflammation can be infectious or non-infectious. As a result of tissue damage, the living organism conceives a chemical signaling cascade that stimulates responses aimed at repairing involved tissues. These signals activate leukocyte chemotaxis from the general circulation to sites of lesion. These activated leukocytes produce cytokines that induce inflammatory responses.

Cytokines are predominantly released from immune cells, including monocytes, macrophages, and lymphocytes. Pro- and anti-inflammatory cytokines facilitate and inhibit inflammation, respectively. Inflammatory cytokines are classified as ILs, colony stimulating factors (CSF), IFNs, TNFs, TGFs, and chemokines, and are produced by cells primarily to recruit leukocytes to the site of infection or injury. Cytokines modulate the immune response to infection

or inflammation and regulate inflammation itself via a complex network of interactions. However, excessive inflammatory cytokine production can lead to tissue damage, hemodynamic changes, organ failure, and ultimately death. Cytokines which activate and promote the inflammatory process (pro-inflammatory) are interleukin 1 (IL1), interleukin 2 (IL2), TNFα and others. Cytokines which inhibit the inflammatory process (anti-inflammatory) are interleukin 10 (IL10), interleukin 1ra (IL1ra), vascular endothelial growth factor (VEGF) and others.

Compelling evidence now exists that inflammation is a major factor in ischemia/reperfusion injury in the kidney. Kidney inflammation contributes to progressive renal injury, which may lead to glomerulonephritis, end-stage renal disease, or acute or chronic kidney disease (CKD) Approximately 10–12% of the population suffers from CKD, and some 50% of elderly patients show signs of kidney dysfunction, which is associated with high morbidity and mortality. Kidney inflammation is most commonly induced by infection, ischemia/reperfusion, in situ immune complex formation/deposition, or complement pathway dysregulation. Renal tubular epithelial cells are likely important promoters of kidney inflammation, secreting a variety of inflammatory cytokines in response to both immune and non-immune factors, and leukocyte infiltration depends on the local presence of these cytokines. Stimuli that can induce kidney injury activate transcription factors (NF-κB or MAPK). These stimuli include cytokines, growth factors, DAMPs, and PAMPs, and metabolic (high glucose, advanced glycosylation end products) and immune mediators.

CKD may be a valid model to illustrate the cytokine network hypothesis. Pro-inflammatory cytokines are counterbalanced at several levels. For example, the secretion of interleukin (IL)-1β is linked to the secretion of the IL-1 receptor antagonist (IL-1RA), which binds the cytokine and prevents its actions. The same mechanism applies to tumor necrosis factor (TNF-α), which is counterbalanced by soluble TNF receptors.

So, a better understanding of how to regulate cytokine pathways would allow for more accurate identification of agent-mediated inflammation and the treatment of kidney inflammatory and noninflammatory diseases. Dysregulation of pro-inflammatory and anti-inflammatory cytokine networks may proceed in parallel and the overall degree of cytokine network disruption may be an important prognostic indicator.

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THE NEGATIVE IMPACT OF XENOBIOTICS ON ION-REGULATING RENAL FUNCTION IN DIFFERENT GROUPS OF ANIMALS

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Prolonged exposure of xenobiotics on human body leads to the formation of chronic diseases, which most often arise on the basis of hereditary predisposition. During long-term admission of xenobiotics, which are not subjected to metabolic changes in the body, it is observed their accumulation (in kidneys, bones, liver), that can cause the occurrence of chronic accumulation diseases. The nephrotoxicity of aluminum salts is one of the components of the universal regenerative-plastic deficiency syndrome, which develops in ecologically damaged regions. Despite the prevalence of aluminum compounds, the question of the effect of aluminum salts on ion-regulating renal function in immature rats.

In experiments on 24 immature rats weighing 0.06-0.10 kg it was investigated the functional state of kidneys, in particular, the ion-regulating function against the background of aluminum salts introduction relative to the control group of animals.

The assessment of the ion-regulating renal function in intact immature rats against the background of aluminum salts introduction, showed that the concentration of sodium ions in the urine increased ($p<0.01$). The excretion of sodium ions tended to increase. The filtration fraction of sodium ions in the conditions of administering aluminum salts in immature rats was characterized by a downward trend compared to the control. The trend toward the growth was recorded for the excretion of sodium ions, standardized by the glomerular filtrate speed. The clearance of water free