



evaluation. Control group included 48 practically healthy persons of relevant age. Gene's nucleotide polymorphism CYP11B2 (-344C/T) was examined by polymerase chain reaction.

TC, LDL-C level in hypertensive patients do not relate directly to polymorphic variants of CYP11B2 (rs1799998) gene. Though, dyslipidemia is more intensively manifested in the T-allele carriers by elevation of TG and atherogenic index (AI) 22.61-56.21% ($p < 0.01$) as much, with lower HDL-C concentration – by 12.23% ($p = 0.043$) and 12.95% ($p = 0.039$), respectively, particularly in men by 25.84 ($p = 0.031$) and 35.76% ($p = 0.042$) higher than in women. CKD evolution in hypertensive patients follows by higher TC, TG and LDL-C that causes an atherogenic index increase (AI) by 13.54% ($p = 0.028$). Polymorphic site of CYP11B2 (rs1799998) gene is associated with TG and AI elevation in general population ($F = 13.98$ and $F = 13.25$; $p < 0.001$), both in women ($F = 22.99$ and $F = 15.21$; $p < 0.001$) and men particularly ($F = 5.09$; $p = 0.018$ and $F = 4.44$; $p = 0.027$) and reduced HDL-C content ($F = 5.28$; $p = 0.007$), especially in men ($F = 9.57$; $p = 0.001$). Furthermore, it associates with WHR increase ($F = 13.09$; $p = 0.003$), especially in the TT-genotype carriers' men ($F = 12.74$; $p < 0.001$).

Thus, polymorphic site of CYP11B2 (rs1799998) gene associates with dyslipidemia: TG and AI elevation, as well as WHR increase in general population, particularly in TT-genotype carriers' men. CKD in hypertensive patients is more related to lipids misbalance, than polymorphic site of CYP11B2 (rs1799998) gene.

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ALTERNATIVE PATHOGENIC APPROACH TO PREVENTION AND TREATMENT OF FLU AND ACUTE RESPIRATORY VIRAL INFECTIONS

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During pandemic coronavirus disease (COVID-19) a number of problems emerged concerning specific diagnostics and possible development of etiotropic and pathogenic treatment. At the same time, common etiopathogenic mechanisms are known to have a special place in occurrence and development of acute respiratory diseases.

The research deals with an alternative pathogenic approach to prevention and treatment of flu and acute respiratory viral infections (ARVI) based on the application of polarized, polychromatic, linear, incoherent, low-energy light (PILER-light), which does not contain ultraviolet and a considerable amount of infrared rays.

Objective of the work was to study changes of the protease-inhibitor system occurring under the action of polarized polychromatic incoherent light (PILER-light) in the body of mice infected by lethal and therapeutic (sub-lethal) doses of A/PR/8/34 (H1N1) flu virus.

A(H1N1/PR/8/34) flu virus was used in the study. The experiments were conducted on albino mice of Balb/c line with the body weight of 13–14 g with the device – a source of polarized polychromatic incoherent light (PILER-light) with the wave length of 400–2000 nm and power 2,4 joule/cm²•min. An active A A/PR/8/34(H1N1) flu virus was obtained, and its lethal dose for mice was determined.

The animals were divided into 4 groups, 10 mice each. The 1st group was infected with a lethal dose of flu A virus through the nose. The 2nd group received the same dose but underwent the course of treatment with PILER-light. The 3rd group was exposed to PILER-light only and the 4th group of mice received saline with diluted flu A virus in it.

The results of the study demonstrate that on the 5th day after being infected all the 100% of animals from the 1st group died. In the 2nd group the animals remained alive on the 14th day after infection. In the 3rd group, where animals were exposed to polarized light only, all of them were active and healthy. All the animals from the 4th group receiving saline remained alive as well. Light therapy of mice infected with a lethal dose of flu A virus (the 2nd group) determined that proteinase activity in the blood serum decreased sharply in comparison with healthy mice (the 3rd group) exposed to light, but it was considerably higher than in the 1st group of mice without treatment.



Therefore, polarized light produces a protective action on the animals with experimental flu infection. In the group of animals infected with a therapeutic (sub-lethal) dose of flu A virus and exposed to the effect of polarized light 80% of animals survived (remained alive on the 15th day after infection). Therefore, polarized polychromatic light can be considered to be an effective therapeutic means in mice infected with influenza virus. It is important that its major protective action is mostly stipulated by the correction of proteinase inhibitor system.

Survival rate in the control 6th group of animals (without PILER-light use) was 50%, and in the 7th group after PILER-light effect – 80%. That is, the reproduction of flu A virus in the body of mice became slow.

Therefore, survival of mice infected with a lethal dose of influenza A virus under PILER-light effect is likely to be the result of an increased content of inhibitor in the lungs compared with that of proteinase activity. This mechanism can be suggested to work in case of diseases caused by other respiratory viruses.

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ASSOCIATION OF GNB3 GENE POLYMORPHISM (rs5443) WITH INDICATORS OF LEFT VENTRICULAR HYPERTROPHY IN PATIENTS WITH ARTERIAL HYPERTENSION CONSIDERING GENDER

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Left ventricular hypertrophy (LVH) is an unfavorable prognostic marker for the development of fatal cardiovascular events, including patients with arterial hypertension (AH). LVH and the geometric structure of the heart are assessed by Left Ventricular Myocardial Mass (LVMM), Left-Ventricular Myocardial Mass Index (LVMMI) and Relative Wall Thickness of Left Ventricle (LV RWT).

The aim of the study was to analyze the relationship of polymorphic variants of the guanine nucleotide binding protein (G-protein) β_3 subunit gene (*GN β_3*) C825T polymorphism (GN β_3 , 825C>T; dbSNP: rs5443) with the indicators of LVMM, LVMMI, LV RWT in patients with primary (essential) hypertension (EH).

A cross-sectional study had been involved 72 patients with EH stage II, 1-3 degrees of blood pressure, high and very high cardiovascular risk. There were 29,16% (21) men, 70,84% (51) women among patients. The average age of patients was $59,87 \pm 7,98$ years. The control group consisted of 48 healthy individuals, comparable in age ($49,13 \pm 6,28$ years) and sex distribution (62,5% – women, 37,5% – men). *GN β_3* C825T polymorphism had investigated by PRL in real time. To establish LVH, all patients had been undergone echocardiography. LVH was calculated by LVMM (according to the Penn Convention) and LVMMI, geometric models – by LV RWT and LVMMI. To evaluate LVH, LVMMI were taken ≥ 115 g/m² in men, ≥ 95 g/m² in women (ESC, ESH 2018).

We have found that men carrying the CC genotype of the *GN β_3* gene LVMM was $297,87 \pm 24,54$ g, LVMMI – $139,01 \pm 17,06$ g/m², LV RWT – $0,47 \pm 0,03$ u; in women – $284,74 \pm 20,04$ g/m², $143,29 \pm 9,22$ g/m² and $0,44 \pm 0,03$ u, respectively. In male patients, TC genotype carriers, we have found that LVMM was $308,68 \pm 33,70$ g ($p_{CC,TC} > 0,05$), LVMMI – $142,75 \pm 15,38$ g/m² ($p_{CC,TC} > 0,05$), LV RWT – $0,45 \pm 0,02$ u ($p_{CC,TC} > 0,05$); in women carrying the TC genotype – $303,68 \pm 15,77$ g ($p_{CC,TC} > 0,05$), $156,98 \pm 12,42$ g/m² ($p_{CC,TC} > 0,05$) and $0,45 \pm 0,03$ u ($p_{CC,TC} > 0,05$), respectively. In patients with EH, carriers of the TT genotype of the *GN β_3* gene, we have estimated that: in men LVMM was $391,47 \pm 31,62$ g ($p_{CC,TC} < 0,05$), LVMMI – $179,82 \pm 10,60$ g/m² ($p_{CC,TC} < 0,05$), LV RWT – $0,45 \pm 0,01$ u ($p_{CC,TC} > 0,05$); in women with TT genotype LVMM $338,25 \pm 16,09$ g ($p_{CC,TC} < 0,05$); LVMMI – $165,41 \pm 10,86$ g/m² ($p_{CC,TC} < 0,05$), LV RWT – $0,45 \pm 0,03$ u.

Thus, the *GN β_3* gene (rs5443) is associated with greater LVH in patients with EH carriers of the TT genotype, especially in men, with LVMMI – by 29,36% and 25,97% ($p_{CC,TC} < 0,05$), slightly less in women – by 15,44% ($p_{CC,TC} < 0,05$) and 5,37% ($p_{TC} > 0,05$), respectively.