Появление сигнала фосфолипидов после ТСМ в крови крыс может быть обусловлено выходом в кровь фосфолипидов мембран разрушенных клеточных структур спинного мозга и/или из мембран эритроцитов, разрушающихся в условиях гипоксии и ацидоза.

Для всех четырех интенсивных линий 1-4 ЯМР $^{31}$P в образцах крови крыс с травмой спинного мозга было характерно смещение в сторону уменьшения резонансных частот. Это означает, что при ТСМ уменьшается pH крови – возникает ацидоз. Уменьшение химсдвига линий 1-4 варьировало от 0.1 до 0.4 ppm. Метод ЯМР позволил детализировать изменение pH: так, смещение линий 1, 2, 4 при травме свидетельствовало о снижении pH внутри эритроцита, а смещение линии 3 об уменьшении pH в плазме крови.

**Выводы.** Таким образом, информация, получаемая методом $^{31}$P ЯМР-спектроскопии, позволяет судить о компенсаторно-приспособительных изменениях после травмы спинного мозга, в частности оценить степень гипоксии, ацидоза и дефосфорилирования АТФ, а сигналы $^{31}$P ЯМР фосфолипидов могут служить индикатором степени разрушения клеточных структур. Сравнение параметров резонансов $^{31}$P ЯМР-спектров эритроцитов у крыс с травмой спинного мозга и интактных животных позволило выявить существенные различия во всех областях спектра, что свидетельствует о значительных сдвигах в уровне и составе фосфоросодержащих соединений при развитии патологического процесса.

*Работа выполнена за счет средств субсидии, выделенной Казанскому федеральному университету для выполнения государственного задания в сфере научной деятельности №17.9783.2017/8.9.*

## CHOLESTEROL LOWERING THERAPY AND LOW DENSITY LIPOPROTEIN PEROXIDATION DURING ATHEROSCLEROSIS

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To study the effect of therapy of patients with CAD by statins or PCSK9 inhibitor on the level of oxidatively modified low-density lipoprotein (LDL) from blood plasma and the activity of erythrocyte Se-containing glutathione peroxidase (GSH-Px).

**Subjects and methods.** Patients with CAD (9-10 men per group) were included in the study, who underwent therapy for a 6-month period including statins - 40 mg/day of pravastatin (group 1) or 0.4 mg/day of cerivastatin (group 2), as well as therapy with the inclusion of the inhibitor PCSK9 - 420 mg/month evolocoumab (group 3) for 1 year. The level of lipohydroperoxides in LDL (LOOH-LDL) was measured in groups 1 and 2 by a modified method using the Fe-xylenolorange



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reagent; the content of oxidatively modified LDL (ox-LDL) in group 3 – using the immunochemical method (Mercodia kits, Sweden). GSH-Px activity in all groups was determined by a modified method with glutathione reductase system using tert-butyl hydroperoxide as a substrate.

**Results.** LOOH-LDL level was significantly increased in groups 1 and 2 (group 2 - in 6-7 times after 3-6 months of therapy) simultaneously with the decrease in LDL cholesterol (LDL-c). In group 2 there was a sharp drop in GSH-Px activity, starting from the 3rd month of therapy. In group 3, when there was a decrease in the concentration of LDL-c, a significant decrease in the level of ox-LDL was observed in the absence of changes in GSH-Px activity.

**Conclusion.** Statins, while effectively reducing the level of LDL-c, simultaneously induce oxidative modification of LDL and decrease GSH-Px activity [1-13]. The PCSK9 inhibitor not only effectively decreases the level of LDL-c, but also reduces the content of ox-LDL without decreasing in GSH-Px activity.

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