the gene *degU*, leading to stabilization of Degu~P protein. It is known, that this mutation leads to a multiple increase in the gene expression level, positively regulated by DegS-DegU system. Our data shows a 10-fold increase in metalloproteinase productivity in the recombinant strain. Thus, Deg-system participates in control of the proteinase synthesis but not only in the regulation of *mprBp* gene expression. The *mprBp* expression in the strain deficient in regulatory protein Spo0A remained at the level with expression in the strain with the complete *spo0A*. A similar pattern we observed in the study of *mprBp* gene expression in strains defective in other spore-specific regulatory proteins (Spo0B, Spo0F, Spo0K, Spo0J, SigF, SigH, SigK). These data indicate that *mprBp* gene expression is free of Spo-regulatory proteins. On this basis, we concluded that the expression of metalloproteinase gene is not correlated with the sporulation.

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Paricalcitol attenuate activity and expression of matrix metalloproteinases in a rat model of renal ischemia-reperfusion injury

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Matrix metalloproteinases (MMPs) are endopeptidases involved in the degradation of extracellular matrix. They have been postulated to have a role in the pathogenesis of ischemia-reperfusion injury (IRI). In the present study, we investigated the effect of paricalcitol, a synthetic vitamin D analog, on MMPs in renal IRI. 21 wistar albino rats were divided into three groups: sham operated, ischemia-reperfusion, and paricalcitol-pretreated. IRI model was induced by bilateral clamping of renal arteries for 45 min followed by 24 h of reperfusion. The analysis of serum creatinine levels and activities/expressions of MMP-2 and -9 were performed after 24 h of IRI. The effects of paricalcitol on activities and expressions of MMP-2 and MMP-9 levels were investigated by gelatin zymography and immunohistochemistry, respectively. The pathological examinations were performed to score tubular damage by light microscopy. Creatinine levels increased significantly in the IRI group. Rats in the paricalcitol-pretreated group showed significant decrease in expressions and activities of MMP-2 and MMP-9 during IRI. Moreover, pathological examinations displayed significantly lower score of tubular damage in paricalcitol-pretreated group. In conclusion, Paricalcitol attenuated IRI by downregulating the expressions and activities of MMP-2 and -9.

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The changes of matrix metalloproteinase 2, 9 activity and hyaluronic acid level in rat's heart and serum under cadmium influence

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The changes in the molecular mechanisms of the extracellular matrix degradation under toxic factors are not well known. The main goal of work was the investigation of the MMP2 and MMP9 activity and hyaluronic acid level in the heart and blood serum under cadmium influence at different doses.

The 18 Wister rats divided to 3 groups were used for the experiment. $CdCl_2x2.5H_2O$ in doses 0.1 μg/kg and 1 μg/kg was given to rats intragastrically in drinking water during 36 days. The rats were decapitated under Isoflurane anesthesia according to ethical rules; the heart was quickly removed. The relative activity [in arbitrary units (au)] of pro- and active forms of MMP9 and MMP2, total protein (TP) and hyaluronic acid levels were calculated.

It was shown that low doses of exogenous cadmium (0.1 μg/kg) lead to reduced activity of pro- and active forms of MMP9 in myocardium (7.3 ± 0.6 au/mgTP and 7.1 ± 0.6 au/mgTP compare to the 9.67 ± 0.4 au/mgTP and 9.7 ± 0.5 au/mgTP in the control rats accordingly) and in serum (0.95 ± 0.2 au/mgTP and 0.35 ± 0.05 au/mgTP compare to the 1.54 ± 0.05 au/mgTP and 1.49 ± 0.05 au/mgTP in the control rats accordingly), but pro-MMP2 activity in heart was increased (14.5 ± 1.6 au/mgTP compare to the 9.8 ± 0.6 au/mgTP in the control rats); level of HA was decreased in both tissues (0.69 ± 0.16 μg/ml and 3.63 ± 0.3 μg/ml compare to the 1.0 ± 0.13 μg/ml and 3.91 ± 0.3 μg/ml in the control rats accordingly). High doses of cadmium (1 μg/kg) caused a reliable increase of both gelatinase activity in the myocardium: MMP2 increased from 9.65 ± 0.4 au/mgTP to 14.1 ± 0.8 au/mgTP, proMMP9 – to 12.6 ± 1.5 au/mgTP, MMP9 – to 15.4 ± 1.6 au/mgTP. HA level was increased in serum (4.28 ± 0.1 μg/ml) and decreased in heart (0.49 ± 0.09 μg/ml).

The results indicate the dose-dependent and tissue-specific effect of cadmium on MMP-depended protein degradation and level of hyaluronic acid.

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IL-33 stimulation down-regulated ADAMTS15 gene and protein in U118 glioblastoma cells

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A disintegrin-like and metalloproteinase domain with thrombospondin-1 repeats (ADAMTS) are a large family of proteoglycanase that show proteolytic activity towards proteoglycans like aggrecan, brevican, neurocan, and versican. Interleukin-33 (IL-33) is an IL-1 cytokine family member that uniquely plays a role as a cytokine and nuclear factor. It is released by necrotic epithelial cells and activated innate immune cells as an alarming danger signal. ADAMTS and IL-33 implicated in brain cancer pathogenesis. We aimed to seek the amount of ADAMTS15 in U118