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DIFFERENTIAL EXPRESSION PATTERN OF THE MRPS18 FAMILY GENES IN BRAIN TUMOR SAMPLES

Background. Brain tumors account for 2%—3% of all malignant neoplasms and 85%—90% of all primary tumors of the central nervous system with the 5-year survival rate of 35%. Additional biomarkers could help refine the molecular profile of brain tumors and prognosis of the disease. **Aim**. To study differential expression patterns of the MRPS18 family genes in tumor tissue and the peripheral blood of patients with brain tumors of various types. **Materials and Methods**. The total RNA was isolated from blood and tumor tissue samples of 27 patients with brain tumors. The quantitative polymerase chain reaction (qPCR) was performed. Also, immunohistochemical (IHC) studies of the MRPS18 family proteins were performed on deparaffinized tissue sections. **Results**. The *MRPS18-1-3* genes were highly expressed at the mRNA level in tumor tissue and the peripheral blood of patients with brain tumors. All 3 genes showed different patterns of expression depending on the tumor type. The highest *MRPS18-1* mRNA expression was detected in glioblastoma (GB) samples in both tumor samples and the peripheral blood. In general, *MRPS18-1* expression was higher in G4 tumors, compared to G2. *MRPS18-3* gene was expressed as higher levels in G2 samples and in embryonic tumors. *MRPS18-2* was expressed in all studied samples, with no regard to the tumor grade or type. The MRPS18-2 IHC staining was detected at high levels in most brain tumors. **Conclusions**. The MRPS18 family genes showed similar patterns of mRNA expression in tissue samples of brain tumors and peripheral blood of patients. The highest levels of *MRPS18-1* mRNA were detected in GB samples, while the highest protein signal was detected for MRPS18-2 in almost all brain tumor samples.

Keywords: MRPS18 family proteins, brain tumors, expression pattern.

Currently, brain tumors account for 2—3% of all malignant neoplasms and 85—90% of all primary tumors of the central nervous system (CNS) [1]. Usually, the 5-year survival rate is quite low for malignant brain tumors and is about 35%. The majority of all brain tumors (approximately 60%) are neuroepithelial neoplasms. There are significant differences in the types of brain tumors in adults and children [2].

Approximately 80% of all brain tumors are gliomas, which originate from glial cells. Gliomas, like

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other tumors, are classified based on their histological characteristics and molecular genetic markers, with the most common subtypes being astrocytoma, oligodendroglioma in adults (adult-type diffuse gliomas), and embryonal CNS tumors in children. Diffuse gliomas in adults are divided into astrocytomas (with *IDH* mutation, Grade 2—4), oligodendrogliomas (with *IDH* mutation and simultaneous deletion of 1p19q, Grade 2, 3), and glioblastomas (GBs, with *IDH*-wild type, Grade 4). Astrocytic gliomas include pilocytic astrocytomas, highly malignant astrocytomas, pleomorphic xanthoastrocytomas, subependymal giant cell astrocytomas, chordoid gliomas, and astroblastomas (MN1-altered) [3].

Pilocytic astrocytoma (PAC) most often occurs in childhood and youth. PACs are classified as Grade 1 tumors, according to the WHO classification [3, 4]. Diffuse astrocytomas (DACs) usually occurring in young people do not show clear boundaries and grow slowly (Grade 2) [3, 5].

Grade 3 diffuse astrocytomas (DACs G3), earlier known as anaplastic astrocytomas (AACs) belong to a group of highly malignant tumors with poor clinical prognosis. GBs that arise from glia are highly aggressive malignant brain tumors. The average life expectancy for GB patients is very low — 12—18 months from the moment of diagnosis [4, 6—8].

Less common are embryonal tumors of the CNS, which are formed in cells remaining after intrauterine development (embryonic cells). Pinealoma, or pineoblastoma (PB) is formed due to the proliferation of various cells of the pineal body — pinealocytes and astrocytes. Its clinical manifestation is similar to medulloblastoma (MB), which also develops from embryonic cells [9]. MB originates from progenitor cells of the developing cerebellum, namely from its outer granular layer [9, 10]. This tumor is characterized by the invasive spread and the ability to metastasize through the cerebrospinal fluid (CSF), forming macrometastases on the surface of the brain and spinal cord.

Meningioma, formed from the structures of the meninges of the brain and spinal cord, is quite common, especially in women aged 40—60 years. Atypical meningioma (AMG), although it is characterized as Grade 2 neoplasm, can be multiple and recurrent.

The molecular mechanisms of malignant cell transformation are extremely complex and individual, so the further research is needed. Such transformation could be caused by the disruption of the important signaling pathways, regulating brain cell growth and differentiation, with the involvement of *SHH, WNT1, PTCH1, PTCH2, SMO, TP53*, and *RB1* genes and/or their products [11—14]. Noteworthy, RB protein function is inhibited by the direct binding with MRPS18-2, one of the proteins of the mitochondrial ribosomal protein S18 (MRPS18) family [15, 16]. In humans, this family contains three members (*MRPS18-1-3*) that arise from one ancestor gene in bacteria [17]. Earlier, we have shown the differential pattern of expression of these genes in glioblastomas [18]. However, there are no data on their expression in other types of brain tumors. Therefore, in the present work we aimed to assess the features of MRPS18 family gene expression at the mRNA and protein levels in human brain tumors in the search for new prognostic markers of the disease.

Materials and Methods

Cohort of patients. In the present work, we have studied 27 surgically resected brain tumor samples and the matched samples of the peripheral blood. The samples were collected at the State Institution "AP Romodanov Institute of Neurosurgery of the National Academy of Medical Sciences of Ukraine (NAMNU)", Kyiv. The diagnosis was established by experienced pathologists from the Department of Neuropathomorphology. The patients did not receive any special treatment prior to surgery. The use of clinical materials was approved by the Ethics Committee of the State Institute "AP Romodanov Institute of Neurosurgery of NAMNU", according to the Declaration of Helsinki from the patients/patients' parents provided the informed consent on the study. All experimental work was carried out according to approved protocols.

The samples of the following tumors were included in the study: pilocytic astrocytoma (PAC, G2, 2 samples), atypical meningioma (AMG, G2, 3 samples), diffuse astrocytoma G2 (DAC-G2, 3 samples) not otherwise specified, (NOS) diffuse astrocytoma G3 NOS (DAC-G3, 5 samples), glioblastoma NOS (GB, G4, 4 samples), embryonal tumor of CNS NOS (EP, G4, 3 samples), pineoblastoma (PB, G4, 1 sample), and medulloblastoma (MB, G4, 6 samples).

Isolation of RNA, synthesis of cDNA, and quantitative (q) PCR. The total RNA from peripheral blood was isolated using the RNeasy Mini Kit (Qiagen Inc., Germany), according to the manufacturer's instructions. For total tumor mRNA extraction, tissue samples were homogenized in liquid nitrogen. Then TRIzol isolation reagent (ThermoFisher Scientific, USA) was added at a ratio of 0.5 mL of buffer per 2 g of tissue. The samples were incubated at room temperature for 10 min. After that, 0.5 mL of chloroform was added for 2—3 min, and the reaction mixture was centrifuged for 20 min at 12,000 rpm. The aqueous phase, which contained mRNA, was transferred to a new test tube, and isopropanol (1/3 of the volume of the aqueous phase) was added. The samples were mixed gently and incubated for 30 min at –20 °C to precipitate the mRNA. After centrifugation for 20 min at 12,000 rpm, the precipitated mRNA was washed twice with 70% ethanol and then dissolved in RNase-free water. The concentration of RNA was estimated using a "Nanodrop ND 1000" spectrophotometer (ThermoFisher Scientific).

The resulting mRNA (final concentration 25 ng/μL) was used for cDNA synthesis, using the RevertAid RT Reverse Transcription Kit (ThermoFisher Scientific, USA), according to the manufacturer's protocol.

Real-time quantitative polymerase chain reaction (qPCR) was used to assess the relative expression of the MRPS18 family genes at the mRNA level. The amplification reaction mixture consisted of 2 μL cDNA, 4 μL 5x HOT FIREPol EvaGreen qPCR Mix (Solis BioDyne, Estonia), 13 μL RNase-free H_2O, and 1 μL of primer mix (reverse and forward, final concentration 25 pM). Primers were the following: for *MRPS18-1* (NM_016067): forward -5'-CAGGTATCCAGCAATGAGG ACC-3', reverse 5'-GCATCCAGTAAAT GGAGA AACAAAC-3'; for *MRPS18-2* (NM_014046): forward 5'-CACAGCGGA CTCGGAAGACATG-3', reverse 5'-GCGCAGACAAATTGCTCCAAGAG-3'; for MRPS18-3 (NM_018135): forward 5'-CATCTGCCGTTGGAACCTTGAAG-3', reverse 5'-CTTGCGGTGTTCTTCCTGG CAT-3'. As an internal control for standardization, a gene encoding TATA-binding protein (*TBP*, NM_003194) was used: forward primer - 5'-TTTCTTGCCAGTC TGGAC-3', reverse 5'-CACGAACCACG GCACTGATT-3'. Relative quantification was used to compare the expression levels of the MRPS18 family genes with the internal control. Two or three reactions (each in triplicate) were run for each gene, so the standard deviation might be calculated. The data were calculated by the $2^{-\Delta\Delta Ct}$ method.

Immunohistochemical (IHC) analysis. The IHC reaction was performed, using the rabbit polyclonal antibodies against the MRPS18-1-3 proteins (Life Technologies, USA) on 5-μm thick tissue sections.

Tissue sections were de-paraffinized in xylene after 10 min of heating at 55 °C. The remaining xylene was washed off with ethanol, and the sections were rehydrated by consecutive washing in phosphate-buffered saline (PBS) with 90%, 70%, 30%, and 0% ethanol. The sections were kept in 2% hydrogen peroxide in methanol for 15 min at RT to reduce the background. To preserve their structure, they were treated with a hot citrate buffer at 92 °C for 20 min with gradual cooling.

Antibodies against the MRPS18-1, -2, and -3 proteins were diluted 1:200 in a blocking buffer (2% bovine serum albumin, 0.2% TWEEN-20, 10% glycerol, and 0.05% NaN_3 in PBS) and were applied to the sections for 1 h. The EnVision system (DAKO, Denmark) was used to visualize the protein signals. Peroxidase activity was determined using DAB after washing the samples in PBS. Nuclei were counterstained with hematoxylin for 1 min, after which sections were dehydrated and mounted in Canadian balsam; then the stained slides were visualized using a Leica microscope.

Statistical analysis. The GraphPad Prism software (version 9, GraphPad Software, La Jolla, USA) was used to evaluate expression, and for assessment of non-parametric data within groups for each gene, the Kruskal — Wallis test was used.

Results and Discussion

Relative expression of the MRPS18 family genes at mRNA levels in brain tumor samples. The relative expression of the MRPS18 family genes at mRNA levels was assessed in 27 brain tumor samples by qPCR. The highest relative expression level was detected for the *MRPS18-1* gene (up to 28 conditional units (c.u.)) in GB samples (Fig. 1, the top panel). The lowest expression was detected in G2 tumors and embryonal tumors. The *MRPS18-2* gene was basically expressed in all tumors, with no difference between tumor types (Fig. 2, the top panel). Only in AMG and PB samples, the mRNA

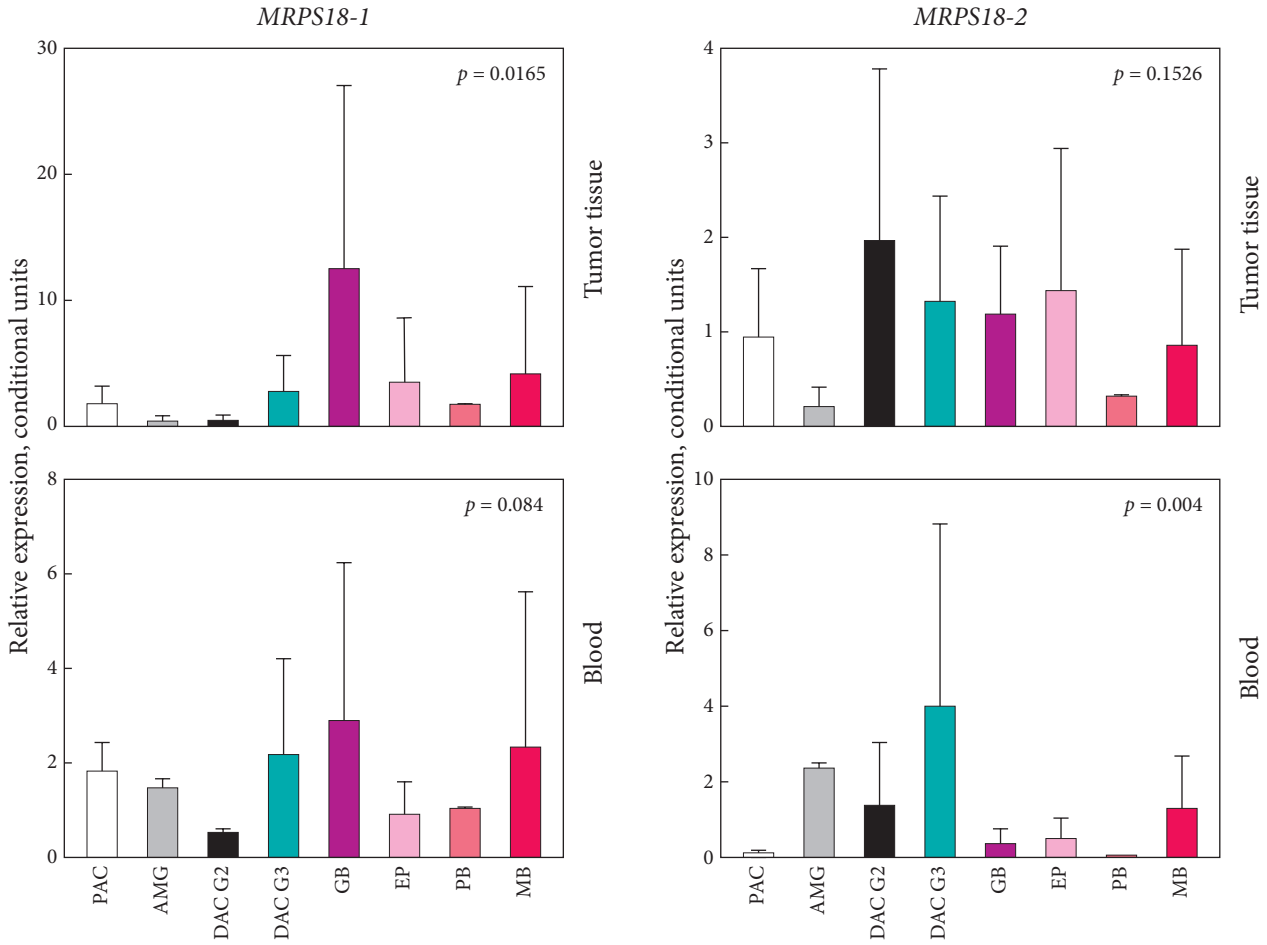

Fig. 1. Expression patterns of the *MRPS18-1* gene at mRNA levels in tumor tissue and peripheral blood samples, assessed by qPCR. The diagram was prepared using GraphPad Prism software; the Kruskal — Wallis test for non-parametric values in groups was applied for each gene. Differences were considered significant at $p \leq 0.05$

Fig. 2. Expression patterns of the *MRPS18-2* gene at mRNA levels in tumor tissue and peripheral blood samples, assessed by qPCR. The diagram was prepared using GraphPad Prism software; the Kruskal — Wallis test for non-parametric values in groups was applied for each gene. Differences were considered significant at $p \leq 0.05$

expression levels of *MRPS18-2* genes were quite low. The relative expression of *MRPS18-3* mRNA was somewhat higher in low-grade tumors such as PAC, AMG, DAC G2, and embryonal tumors (Fig. 3, top panel).

Earlier, we have shown that *MRPS18-2* gene is expressed at quite high level in many tumors, in particular, endometrial, breast, prostate tumors, glioblastoma, several lymphomas (summarized in [19]), and medulloblastoma [20].

Regarding the published data on *MRPS18-3* gene, it has been shown that this gene is expressed at high level in breast tumors [21]. Recently, bioinformatic analysis also revealed the high MRPS18-1 expression in breast cancer [22].

Detection of the extracellular mRNA of the MRPS18 family genes in the peripheral blood of patients with brain tumors. A relative amount of the extracellular mRNA of the MRPS18 family genes in the samples of the peripheral blood of 27 patients with brain tumors was assessed by qPCR.

Earlier, we have demonstrated the possibility of isolating a sufficient amount of extracellular mRNA from the blood serum of cancer patients for the analysis of the gene expression patterns in the blood serum [23]. It is possible to assess the extracellular mRNA levels, especially, when the experimental data were normalized to the specific control, the TBP gene expression.

The relative expressions of the *MRPS18-2* and *MRPS18-3* genes in the blood were quite different from those in the tumor tissues, with only few exceptions. In particular, similar patterns of the *MRPS18-3* expression were detected in DAC G3

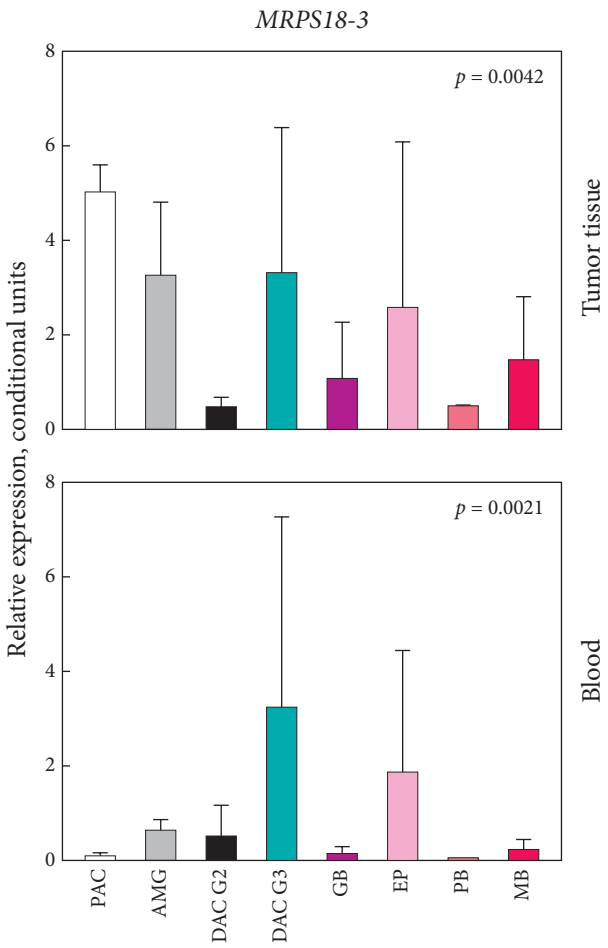

Fig. 3. Expression patterns of the *MRPS18-3* gene at mRNA levels in tumor tissue and peripheral blood samples, assessed by qPCR. The diagram was prepared using GraphPad Prism software; the Kruskal — Wallis test for non-parametric values in groups was applied for each gene. Differences were considered significant at $p \leq 0.05$

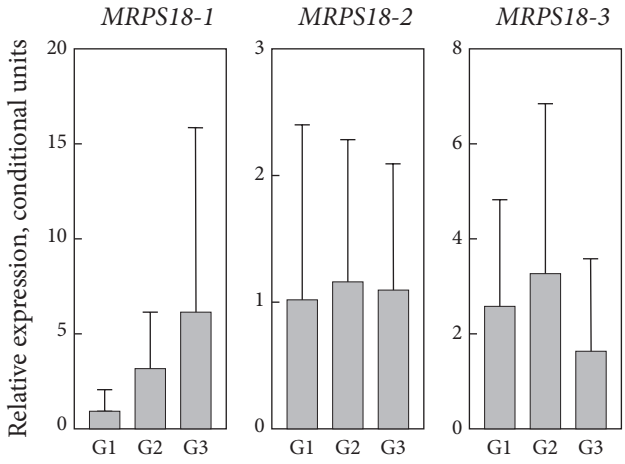

Fig. 4. Expression patterns of the MRPS18 family genes at mRNA levels in tumor tissue, depending on the tumor grade. The diagram was prepared using GraphPad Prism software; the Kruskal — Wallis test for non-parametric values in groups was applied for each gene. Differences were considered significant at $p \leq 0.05$

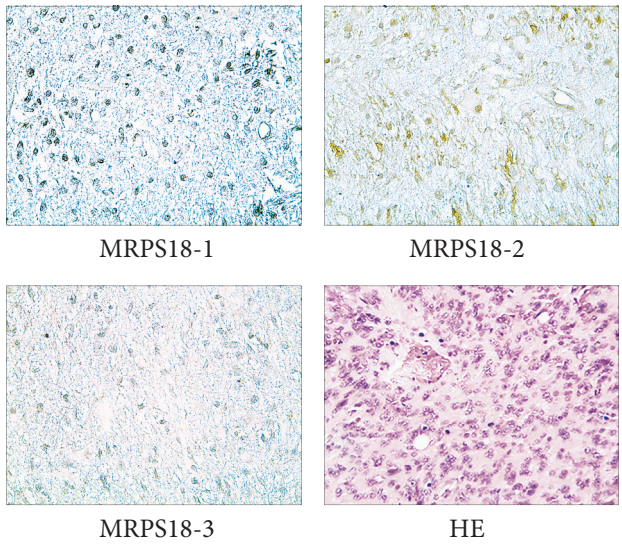

Fig. 5. An example of IHC staining of the MRPS18 family proteins in glioblastoma. Notice strong protein signals of MRPS18-2

and PB, and of MRPS18-2 — in DAC G3 and MB (Figs. 2 and 3, bottom panels). For the *MRPS18-1* gene, the patterns of relative expression were quite similar in both tumor tissue and blood samples (Fig. 1, bottom panel). However, in blood, the expression values were about 4-fold lower.

Next, the mRNA expression levels were analyzed, depending on the tumor grade. No significant differences were found ($p = 0.2212$ for *MRPS18-1*, $p = 0.7521$ for *MRPS18-2*, and $p = 0.3357$ for *MRPS18-3*). However, we detected a clear trend to increasing the *MRPS18-1* expression levels upon tumor progression and the opposite trend for *MRPS18-3*, i.e., in more advanced tumors, the *MRPS18-3* mRNA levels were decreased (Fig. 4). *MRPS18-2* was expressed at approximately similar levels in all studied cases, and we have observed such a pattern earlier in the study of hepatocellular carcinomas, lymphomas, and glioblastomas [19].

Expression patterns of the MRPS18 family genes at the protein level in brain tumor samples. The protein expression showed a slightly different picture. An example of IHC staining of MRPS18 family proteins in glioblastoma is given in Fig. 5 demonstrating that the MRPS18-2 IHC staining intensity was quite high in comparison with the MRPS18-1 and MRPS18-3 expression levels. Noteworthy, in the majority of studied tumor tissues,

the cytoplasmic IHC staining of MRPS18-2 was high, independently of the tumor type. We have observed such an expression pattern of MRPS18-2 earlier in hepatocellular carcinoma samples [19].

It could be hypothesized that the high intensity of the MRPS18-2 IHC staining may be explained by different half-life of the MRPS18 family proteins. We have obtained the preliminary data that the MRPS18-2 protein is quite stable with a half-life of more than 2 h (unpublished data).

In conclusion, the highest relative expression levels were observed for the *MRPS18-1* gene in GB tissue samples and the peripheral blood of patients with GB. It was a trend for increased *MRPS18-1* expression in tumors of a higher grade (less differentiated). The *MRPS18-2* gene was expressed in all tumors, with basically no difference between tumor types. The relative expression of *MRPS18-3* mRNA was somewhat higher in low-grade tumors and embryonal tumors. Moreover, the extracellular mRNA of all three genes of the MRPS18 family can be detected in the blood sera of patients demonstrating in some cases similar patterns of mRNA expression in brain tumor tissue samples and in the patient's peripheral blood. Additional studies on a larger cohort of patients should be provided to assess the putative prognostic value of the MRPS18 family genes in patients with different forms of brain tumors.

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Authors contribution

LK, AS, and SK carried out the immunofluorescence staining and performed qPCR. EK, LK, and OM performed a statistical analysis. TM and VR collected tumor tissue and blood samples. LK, TM, and EK conceived and designed the study. VR, TM, and EK coordinated this study. LK and EK drafted the manuscript. All authors read, edited, and approved the final manuscript.

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ДИФЕРЕНЦІЙНИЙ ПАТЕРН ЕКСПРЕСІЇ ГЕНІВ РОДИНИ MRPS18 У ЗРАЗКАХ ПУХЛИН ГОЛОВНОГО МОЗКУ

На даний момент пухлини головного мозку становлять 2—3% від усіх злоякісних новоутворень і 85—90% від усіх первинних пухлин центральної нервової системи. Зазвичай п'ятирічна виживаність при злоякісних пухлинах мозку досить низька і становить близько 35%. Необхідно знайти додаткові біомаркери, які б допомогли уточнити молекулярний профіль пухлин головного мозку та спрогнозувати перебіг захворювання. **Мета**: вивчити патерн експресії генів родини MRPS18 в пухлинній тканині та периферичній крові пацієнтів з пухлинами головного мозку різного типу як потенційних прогностичних біомаркерів. М**атеріали та методи**. Тотальну РНК виділяли із зразків крові та пухлинної тканини 27 пацієнтів із пухлинами головного мозку. Проводили кількісну полімеразну ланцюгову реакцію (кПЛР) та імуногістохімічні дослідження білків родини MRPS18 на депарафінованих зрізах тканин. Дослідження було доповнено статистичним аналізом. **Результати**. Гени *MRPS181-3* були високо експресовані на рівні мРНК у пухлинній тканині та периферичній крові пацієнтів з пухлинами головного мозку. Усі три гени показали різні моделі експресії, залежно від типу пухлини головного мозку. Найвищу експресію мРНК *MRPS18-1* виявлено у зразках гліобластоми — як у пухлині, так і в периферичній крові. Загалом експресія *MRPS18-1* була вищою в пухлинах G4 порівняно з G2. *MRPS18-3* експресувався на вищих рівнях у зразках G2 і в ембріональних пухлинах. *MRPS18-2* експресувався у всіх досліджуваних зразках, незалежно від типу пухлини. Сигнал протеїна MRPS18-2 виявлено на високих рівнях у більшості пухлин головного мозку. **Висновки**. Гени родини MRPS18 показали подібний патерн експресії мРНК у зразках тканин пухлини мозку та периферичної крові пацієнтів. Найвищі рівні мРНК *MRPS18-1* виявлено в зразках гліобластоми, тоді як найвищий білковий сигнал гена M*RPS18-2* — майже у всіх зразках пухлин головного мозку. Оцінка рівня експресії гена *MRPS18-1* у периферичній крові може бути використана як потенційний прогностичний маркер прогресії пухлини. Такі дослідження треба проводити і далі, на більшій когорті пацієнтів з пухлинами головного мозку.

Ключові слова: білки родини MRPS18, пухлини мозку, патерн експресії.