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SHORT COMMUNICATION

BIOINFORMATIC IDENTIFICATION OF PROTEINS WITH VARYING LEVELS OF POST-TRANSLATIONAL MODIFICATIONS IN A MODEL OF ATHEROGENESIS IN MICE

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Mass spectrometric data obtained using a model of tandem carotid artery stenosis in mice with unstable and stable atherosclerosis were analyzed to identify differences in the level of post-translational modifications (PTMs) of proteins. The original proteomic data obtained by Chen et al. [DOI: 10.1038/s42003-023-04641-4] and deposited in the PRIDE repository (identifier PXD030857) were used. Based on results of the bioinformatic analysis, 12 proteins with PTMs (methylation, acetylation, and phosphorylation) were selected; comparison of healthy and atherosclerotic vascular sections showed that the selected proteins were characterized by significant changes in the level of individual modified peptides. According to the literature data, all 12 proteins are involved in the process of atherogenesis. Our study thus revealed putative points of regulation of the atherogenesis processes at the PTM level.

Keywords: post-translational modifications; atherosclerosis; bioinformatics

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INTRODUCTION

After synthesis on ribosomes, proteins undergo covalent chemical changes known as post-translational modifications (PTMs). These modifications have a significant impact on protein features, regulating such important molecular functions as enzymatic activity, maintenance of the structure of cells and extracellular components, formation of various signaling pathways, and immune defense of the body [1, 2]. PTMs are involved in many biochemical processes in the body, and any changes in them can be both a cause and a consequence of diseases development; they also act as a protective factor or a risk factor in the development of pathological processes [3, 4]. The main groups of diseases affected by PTM disorders include neurodegenerative, cardiovascular diseases, and cancer [5], and the modifications themselves are increasingly considered as markers of various groups of pathologies [3, 6]. However, both PTMs in particular and modified and unmodified proteins in general do not have sufficient pathogenetic specificity as markers of certain diseases, and therefore researchers consider them in combination with other candidate markers, taking into consideration not only the fact of PTM/protein detection, but also their quantitative changes [7]. Analysis of quantitative changes in PTMs, such as phosphorylation, methylation, or glycosylation, in combination with changes in the protein content, provides information on the functioning of cellular pathways, regulation of protein functions and cellular responses to various stimuli [8]. In this context, atherosclerosis is a classic example of how different cell types interact and influence each other, contributing to the development of atherosclerotic plaques and disease progression, and PTMs can increase or decrease their functional activity [9].

The liquid chromatography tandem mass spectrometry (LC-MS/MS) method is the most versatile and effective method for PTM analysis. LC-MS/MS provides information without prior knowledge of a specific modification site and also allows obtaining quantitative data for each specific ion even without the use of isotopic labels [10].

In this study we have analyzed a dataset deposited in the PRIDE repository [11] with the identifier PXD030857. The authors of the original work [12] used a mouse model of tandem carotid artery stenosis (TS), reproducing the mechanism of instability/rupture of atherosclerotic plaques in humans. Using quantitative proteomics methods, the authors [12] revealed differences in the nature of unstable and stable atherosclerosis and identified protein markers of these conditions, as well as identified key proteins and signaling pathway networks that could be used as a preclinical tool for the development and testing of plaque-stabilizing drugs. Although peptides with N-terminal acetylation were taken into account in the analysis, quantitative changes were considered only for whole proteins. The authors did not take into account other possible physiological PTM. In this work, we have tried to analyze the initial data of the work [12] in order to search for proteins with different levels of PTM in the experiment and control.

MATERIALS AND METHODS

The description of the experimental part on the use of laboratory mice, the creation of a model of carotid artery TS for the activation of the atherogenesis process, sample preparation, and proteomic analysis of the obtained samples are given in detail in the original work [12]. Briefly, we have analyzed the data obtained during the proteomic analysis of vascular segments representing unstable atherosclerosis (right carotid artery — unstable plaque), stable atherosclerosis (aortic arch — stable plaque) and a healthy artery (left carotid artery without plaques). Vascular segments from 15 mice were pooled into three groups depending on the plaque type (U — unstable; S — stable) or their absence (H — healthy artery); then 8 samples were selected from each group of segments for protein extraction, gel electrophoresis, in-gel trypsin digestion and label-free quantitative proteomic LC-MS/MS analysis [12].

In our work, peptide identification based on raw files was performed again using the PEAKS-X Pro software [13]. The search was performed by the amino acid sequences of mouse proteins (*Mus musculus*, UniProtKB/Swiss-Prot release 2024_03, 17823 records [14]) using the following search parameters: the cleavage enzyme was trypsin; the peptide mass tolerance was 10 ppm; the fragment mass tolerance was 0.02 Da; the max missed trypsin cleavage sites cleavage was set as 2; the number of variable modifications per peptide was 2 (peptides with a single modification under study were considered in the final analysis). Carbamidomethylation of cysteine was chosen as a fixed modification, and the variable modifications included oxidation of methionine, acetylation of lysine and the N-terminal peptide of the protein, methylation of aspartic and glutamic acid residues, lysine, arginine and serine, phosphorylation of serine, threonine and tyrosine, and deamination of asparagine and glutamine. All options for additional filtering and formation of chimeric spectra were disabled. Modifications of the lysine residue were considered only if the modified lysine was not the last residue in the peptide. The false discovery rate (FDR) for the final selection of identified peptides was 0.1%. Identification was performed for each sample independently.

Using chromatographic data from raw files, the entire space of primary ions was aligned and the area under the peak for each of the primary ions was normalized (Normalized abundance, NA) using the Progenesis LC-MS program [15]. The peptide identification data were imported into the Progenesis LC-MS program project, combining the alignment results with the peptide identification results for subsequent comparison of the quantitative data of the vascular segment samples.

Analysis of significance of changes in the level of peptides with PTM, the parameters of the analysis of variance (ANOVA) performed by the computational algorithms implemented in the Progenesis LC-MS program were evaluated. The change in the level of modified peptides was considered significant at Anova p-value < 0.05 and Max fold change ≥ 2. The max fold change is a measure that describes the changes in the mean NA value between the control (H) and experimental groups (U, S); for example, for two values of NA_H and NA_U, the range of change in NA_U relative to NA_H is NA_U/NA_H.

RESULTS AND DISCUSSION

A total of 165,998 primary ions were selected, of which 9,143 ions were identified with sequences of 4,526 peptides for 839 proteins. The final selection was made for proteins for which at least 5 peptides were used for quantitative determination by the Progenesis LC-MS program. The full set of data on the alignment of the identified ions is presented in the Supplementary Materials.

Table 1 shows identified peptides with PTMs, corresponding to the conditions of Anova p-value < 0.05 and the max fold change ≥ 2 during evaluation of the NA mean differences between experimental groups and the control. Peptides with methylation of aspartic and glutamic acid residues, tyrosine phosphorylation were not identified under the given selection parameters. Peptides with deamination of asparagine and glutamine residues were not considered significant, since the deamination process might depend on the conditions of sample preparation and mass spectrometric determination. For one of the 13 peptides (YIQVVYLHNNNISAVGQNDFCR, decorin), no significant quantitative changes were noted relative to the total protein content. For the others, the change in the average NA of the peptide between the experiment and the control differed from the same parameter for the protein by at least 1.5 times. The peptide DQSILCTGESGAGK (myosin-11) with phosphorylation was the only one for which an increase in quantity was noted in the experiment, while the content of the protein itself (myosin-11) decreased.

All the proteins listed in the Table 1 are involved in the atherogenesis process. For example, transgelin-2 (Q9WVA4) modulates actin-myosin interaction, maintains the stability of actin filaments, regulates muscle contractility and cell migration; a decrease in the transgelin level has been described in vascular diseases, including atherosclerosis [16, 17]. Destrin (Q9R0P5) cleaves actin, inhibits the differentiation of smooth muscle cells, reduces their number and causes impairments in proliferation and migration [18]. Myosin-9 (Q8VDD5) is an actin-binding protein involved in pathological processes associated with the transmission of cellular signals and, consequently, with cell division, endo- and exocytosis,

Table 1. Identified peptides with PTM corresponding to the conditions of Anova p-value < 0.05 and the max fold change ≥ 2 during evaluation of the NA mean differences between experimental groups and the control

#	Protein ID (UniProt)	Protein name (UniProt)	Sequence of modified peptide	PTM position in the peptide sequence	Anova p-value of the peptide	Max fold change of the peptide	Group with the highest and lowest NA mean value of the peptide		Max fold change of the protein	Group with the highest and lowest NA mean value of the protein	
1	Q9WVA4	Transgelin-2	ANRGPSYGLSR	[1] A(+42.01)	0.0002	3.5	H	U	1.5	H	U
2	Q9R0P5	Destrin	ASGVQVADEVCR	[1] A(+42.01)	1.36E-009	4.4	H	U	1.6	H	U
3	Q8VDD5	Myosin-9	AQQAADKYLYVDKNFINNPLAQADWAAK	[1] A(+42.01)	0.0001	2.7	S	H	1.1	S	U
4	Q8BTM8	Filamin-A	IVSPSGAAVPCKVEPGLGADNSVVR	[12] K(+14.02)	0.0010	3.3	H	U	2.1	H	U
5	Q8BTM8	Filamin-A	LKPGAPLRPK	[2] K(+42.01)	1.95E-007	4.7	S	U	2.1	H	U
6	Q08091	Calponin-1	SSAHFNR	[1] S(+42.01)	1.53E-005	3.7	H	U	2.1	H	U
7	Q05793	Basement membrane-specific heparan sulfate proteoglycan core protein	QVHEGRTVR	[6] R(+14.02)	6.60E-006	2.1	S	U	1.2	S	U
8	P60710	Actin. cytoplasmic 1	DDDIAALVVDNGSGMCK	[1] D(+42.01)	0.0126	3.7	U	H	1.0	S	H
9	P37804	Transgelin	ANKGPSYGMSR	[1] A(+42.01)	0.0002	5.5	H	S	2.0	H	U
10	P31428	Dipeptidase 1	THTNIPK	[3] T(+79.97)	0.0310	3.2	S	U	1.3	H	U
11	P28654	Decorin	YIQVVYLHNNNISAVGQNDFCR	[22] R(+14.02)	0.0060	2.0	H	S	2.3	H	S
12	P21956	Lactadherin	GPCSPNPCYNDAK	[4] S(+14.02)	0.0021	3.1	S	U	1.9	S	U
13	O08638	Myosin-11	DQSILCTGESGAGK	[3] S(+79.97)	0.0001	3.0	U	H	2.1	H	U

cellular adhesion, migration, as well as with the inflammatory response; it is a biomarker of early atherosclerotic lesions [19, 20]. Filamin-A (Q8BTM8) is an actin-binding protein that stabilizes actin networks and integrates them with cell membranes, providing cell motility; it participates in cellular signaling and transcription, determining the functions of vascular cells, and is characterized by an increased level of expression in macrophages, which are involved in the development of atherosclerosis [21]. Calponin-1 (Q08091) regulates actin-activated myosin motor activity (regulation of smooth muscle contractility); it controls proliferation and migration of vascular smooth muscle cells, inhibits calponin, slowing down atherogenesis [22]. A specific heparan sulfate proteoglycan (Q05793) is an important component of basement membranes that promotes atherosclerotic plaque formation; it plays an important role in maintaining endothelial barrier functioning, vascular homeostasis, and inhibiting smooth muscle cells proliferation [23]. Cytoplasmic actin 1 (P60710) is involved in the formation of cell structure and integrity; its level is decreased in patients with carotid plaque ruptures [23]. The cytoskeletal protein transgelin (P37804) is expressed exclusively and in high amounts in visceral and vascular smooth muscle cells; it modulates the phenotype of vascular smooth muscle cells during atherogenesis [18, 24]. Dipeptidase 1 (P31428) has been detected in the adventitia of all arteries; it serves as a marker of adventitial fibroblasts [25]. Decorin (P28654) is a rate-limiting enzyme of the polyol pathway, involved in aldehyde detoxification; it has proinflammatory functions. It plays a role in downregulation of immunosuppressive TGFβ and autophagy, which may be important for the development of an inflammatory environment during atherosclerotic plaque formations [26]. Lactadherin (P21956) is a multifunctional glycoprotein involved in many biological and physiological processes, including phagocytosis, angiogenesis, atherosclerosis, tissue remodeling, and regulation of hemostasis. Impairment of lactadherin signaling contributes to aging processes leading to atherosclerosis and neurodegeneration; this protein is considered as a therapeutic target for the treatment of atherosclerosis by preventing plaque formation [27]. Myosin-11 (O08638) is expressed in the medial layer of arteries in human atherosclerotic lesions, where the level of apoptosis is increased. A significant relationship was found between the level of myosin-11 and the presence of multiple atherosclerotic lesions; when a significant number of smooth muscle cells are damaged, both in the aorta and in other arteries, the concentration of circulating myosin-11 may increase [28, 29].

Thus, our analysis of previously published raw data [12] resulted in identification of putative points of regulation of atherogenesis processes at the PTM level. The results obtained require additional verification by more accurate methods than the label-free method of quantitative proteomic analysis.

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COMPLIANCE WITH ETHICAL STANDARDS

This article does not contain any research involving humans or the use of animals as objects.

CONFLICT OF INTEREST

The authors declare conflicts of interest.

Supplementary materials are available in the electronic version at the journal site (pbmc.ibmc.msk.ru).

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БИОИНФОРМАТИЧЕСКАЯ ИДЕНТИФИКАЦИЯ БЕЛКОВ С МЕНЯЮЩИМСЯ УРОВНЕМ ПОСТТРАНСЛЯЦИОННЫХ МОДИФИКАЦИЙ ПРИ МОДЕЛИРОВАНИИ ПРОЦЕССА АТЕРОГЕНЕЗА У МЫШЕЙ

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Проанализированы масс-спектрометрические данные, полученные в эксперименте с использованием модели тандемного стеноза сонных артерий у мышей при нестабильной и стабильной форме атеросклероза, для выявления различий в уровне посттрансляционных модификаций (ПТМ) белков. Исходные протеомные данные получены Chen и соавт. [DOI: 10.1038/s42003-023-04641-4] и депонированы в репозитории PRIDE (идентификатор PXD030857). По результатам анализа отобраны 12 белков с ПТМ (метилирование, ацетилирование и фосфорилирование), имеющих существенные изменения в уровне отдельных модифицированных пептидов при сравнении здоровых и поражённых атеросклерозом участков сосудов. Согласно литературным данным, все 12 белков вовлечены в процесс атерогенеза. Проведённое исследование позволило выявить возможные точки регуляции процессов атерогенеза на уровне ПТМ.

Полный текст статьи на русском языке доступен на сайте журнала (http://pbmc.ibmc.msk.ru).

Ключевые слова: посттрансляционные модификации; атеросклероз; биоинформатика

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