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Association of desmin expression with tumour size in colorectal carcinomas

Andrey Filin*

PhD in Medical Sciences, Associate Professor
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0000-0003-1670-3694

Elena Priputnevich

Medical Resident
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0000-0002-7763-9932

Viktoria Shishkina

PhD in Medical Sciences, Associate Professor
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0000-0001-9185-4578

Abstract. Desmin, which was expressed by muscle cells, myofibroblasts and certain pathological cells, remained poorly investigated in the context of the morphogenesis of colorectal cancer – the third most common oncological disease in the world. The aim of the study was to examine the characteristics of desmin expression in colorectal carcinomas and to compare the intensity of this expression with tumour nodule size. During the study, 24 samples of malignant large-intestinal tumours were subjected to histological examination and were stained using the immunohistochemical method with mouse monoclonal antibodies against desmin. Desmin expression was identified in all analysed samples. Desmin was expressed both within tumour structures and in the surrounding stromal tissue of the organ. Quantitative heterogeneity of desmin expression was revealed, ranging from extensive to scarce across different samples. When the volumetric density of desmin was compared with tumour nodule area, it was found that the overall amount of expressed desmin was statistically significantly higher in samples with large tumour nodules. In addition, a tendency was observed towards an increase in the amount of desmin localised within tumour structures with increasing tumour area. The study also included a comparison of the presence of desmin in the tumour with the size of the tumour conglomerate. It was established that desmin was present within the tumour considerably more often in large tumour nodules than in samples with small tumours. The findings obtained in the course of the study indicated a potential possibility of using desmin to assess the risk of tumour progression, as well as its potential use as a prognostic marker

Keywords: tumour volume; intermediate filament protein; stroma; immunohistochemical method; tumour nodule

INTRODUCTION

Colorectal cancer remained one of the most widespread and socially significant forms of malignant neoplasms, occupying leading positions in both morbidity and mortality. Despite substantial progress in diagnosis and treatment, the biological mechanisms determining tumour morphogenesis and progression continued to be actively investigated. Particular interest was directed towards molecular and morphological markers ca-

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**Corresponding author*

pable of reflecting the features of tumour growth and its invasive potential. One such marker was desmin, an intermediate filament protein that was expressed in muscle cells and in a number of tumour structures, for which new patterns of expression continued to be identified in epithelial neoplasms, including colorectal cancer.

According to F. Bray *et al.* [1], colorectal cancer was the third most common malignant neoplasm, surpassed only by lung cancer and breast cancer, with 1,926,118 new cases registered in 2022, accounting for 9.6% of all forms. In addition, mortality from colorectal cancer was among the highest: in 2022 this disease caused the deaths of 903,859 individuals, representing 9.3% of all cancer-related deaths, which placed it in second position among all malignant neoplasms. Colorectal cancer occurred with nearly equal frequency in men and women. The mean age at diagnosis was 68 years for men and 72 years for women.

Colorectal cancer was most frequently diagnosed at an age above 50 years. Opinions regarding trends towards increasing or decreasing incidence of large-intestinal cancer remained somewhat contradictory. According to E. Morgan *et al.* [2], by 2040 the number of newly diagnosed cases of colorectal cancer would reach 3.2 million, and the number of deaths would rise to 1.6 million. At the same time, E.M. Stoffel & C.C. Murphy [3] reported an overall trend towards a decline in the incidence of large-intestinal cancer in the general population, while emphasising another issue: colorectal cancer had become more common among younger individuals. According to their data, the incidence of colorectal cancer in the general population had decreased from 1990 to the present, yet the number of registered cases in young patients had almost doubled, a trend particularly evident in the 40-49-year age group. This trend was further confirmed by the study of L.-F. Hu *et al.* [4], which reported an annual increase of 2% in new colorectal cancer cases in patients younger than 50 years. The same study highlighted specific clinical features in this age group, such as a predominance of poorly differentiated tumours, left-sided localisation of the process and more frequent diagnosis at advanced stages. The issue of rising incidence of colorectal cancer was also addressed in the study of S.G. Patel *et al.* [5], which noted an annual increase in large-intestinal cancer among younger patients and drew attention to the potential causes underlying this trend. The annual rise in colorectal cancer incidence among young individuals indicated the relevance of this problem and the necessity of its detailed investigation.

In the investigation of colorectal cancer, considerable attention was directed towards various clinical and morphological indicators, such as tumour nodule size. In malignant neoplasms of different localisations, tumour nodule size was most often regarded as a poor prognostic factor. Tumour size frequently correlated with the character and rate of its growth. Large tumours generally demonstrated more aggressive behaviour and a greater potential for invasion and metastasis. Opinions regarding the prognostic role of tumour nodule size in colorectal cancer remained contradictory. According to the study by Y. Meng *et al.* [6], no statistically significant correlation was identified between tumour nodule size and patient survival. For small tumours (less than 3.5 cm) and large tumours (greater than 5 cm), patient prognosis was equally poor, whereas tumours of intermediate size (3.5-5 cm) were associated with better survival outcomes. However, these findings were contradicted by the study of S. Saha *et al.* [7], which demonstrated a correlation between tumour size and patient survival in colorectal cancer. When tumour nodules exceeded 6 cm, patient survival was significantly worse than in cases with smaller tumours.

Desmin was an intermediate filament protein localised in sarcomeres near the Z-line. Since its discovery, various functions had been attributed to desmin, ranging from the regulation of myofibrillogenesis, the establishment of mitochondrial positioning within the cell and participation in their functioning, to determining the resistance of muscle cells to stretching [8]. Desmin attached the Z-disc to the cytoplasmic portion of the muscle-cell membrane, which ensured the preservation of structural and mechanical integrity of the muscle cell during contraction. Desmin was expressed both directly by muscle cells and by tumour cells that had acquired muscle-cell properties [9].

Desmin expression was most frequently examined in tumours arising from muscle tissue; however, in recent years the number of studies addressing the role of desmin in the morphogenesis of tumours of epithelial origin had increased. These were most commonly tumours of the gastrointestinal tract. Although the number of studies on this topic had grown annually, the role of desmin in the morphogenesis of colorectal cancer remained far from fully understood. Most often, desmin expression in colorectal carcinomas had been compared with such clinical and morphological indicators as tumour differentiation grade, disease stage and depth of invasion. In the study by G. Arentz *et al.* [10], desmin was found to be expressed more extensively at advanced stages of the disease than at the first stage. In the study of M. Yoshida *et al.* [11], the authors concluded that desmin was effective for assessing the degree of tumour invasion in colorectal cancer. All the above indicated the relevance of the topic and the necessity of investigating the role of desmin in the morphogenesis of colorectal cancer.

The aim of the study was to investigate the morphological patterns of desmin expression in colorectal cancer and to relate these patterns to a significant morphological indicator, namely tumour nodule size.

MATERIALS AND METHODS

A histological examination was carried out on material from 24 patients with diagnosed colorectal cancer. Material selection was performed at the Voronezh Regional Pathoanatomical Bureau (Russia, Voronezh). Further processing and analysis of the material were conducted at the Research Institute of Experimental Biology and Medicine (Russia, Voronezh), as well as at the Departments of Pathological Anatomy and Histology of N.N. Burdenko Voronezh State Medical University (Russia, Voronezh). The study was performed in accordance with the ethical standards of the Declaration of Helsinki [12]. The study protocol was approved by the local ethics committee of N.N. Burdenko Voronezh State Medical University (Protocol No. 6 of 14.11.2022). All participants were provided with written information about the aims and procedures of the study, and voluntary informed consent was obtained from each participant prior to inclusion. Material collection was conducted during 2023-2025. The analysed samples were represented by segments of the large intestine removed during surgical interventions. For this study, only tumours localised in the sigmoid colon were selected. Exclusively tumours not exposed to neoadjuvant treatment were used, in order to exclude the potential influence of treatment-induced pathomorphosis on the results. According to histological type, all samples belonged to adenocarcinomas of various differentiation grades: 12 well-differentiated (Grade 1), 9 moderately differentiated (Grade 2) and 3 poorly differentiated (Grade 3) adenocarcinomas. To assess tumour size, its area on the gross specimen was measured. Each sample was photographed in a single plane together with a reference scale ruler. The tumour nodule area in square centimetres (cm^2) was calculated by a planimetric method using Adobe Photoshop 2024, based on the known scale. To detect desmin, the immunohistochemical method was applied to paraffin sections using monoclonal antibodies against desmin. Histological material was fixed in 10% neutral buffered formalin immediately after sampling. Fixation was performed at room temperature for 3 days, after which standard tissue processing with paraffin embedding was carried out [13]. For histological examination of desmin (the cytoskeletal protein) expression, thin 2-μm paraffin sections were prepared and subjected to standard processing. Immunohistochemical detection was performed manually according to a standard protocol [14]. Samples of skeletal muscle tissue served as the positive control, whereas lymph-node sections served as the negative control. According to the protocol, primary monoclonal rabbit antibodies Anti-Desmin antibody [Y66] (catalogue number #ab32362, dilution 1:2000, manufacturer: Abcam, United Kingdom) were applied and incubated for 12 hours, after which secondary Goat anti-rabbit IgG H&L (HRP), conjugated with horseradish peroxidase (catalogue number #ab205718, dilution 1:2000, manufacturer: Abcam, United Kingdom), were applied. Nuclear contrast was achieved by counterstaining with Mayer's haematoxylin. Desmin expression intensity and its predominant localisation were assessed: in the stroma or in close proximity to tumour structures (hereafter referred to as in the tumour or in tumour structures). To quantify desmin expression, its volumetric density (Ss) was calculated in each sample by determining the ratio of the area occupied by desmin in a given region to the total section area. In addition, the mere presence or absence of desmin expression directly within tumour structures was evaluated. For each sample, ten random fields of view were selected. Images were captured using a Carl Zeiss Primo Star microscope (Germany), 40×/0.45 objective. Volumetric density was calculated using Adobe Photoshop 2024. Counting was performed by two independent observers. For statistical analysis of the obtained results, Microsoft Excel 2024 was used. The normality of distribution of quantitative data was assessed by graphical analysis (Q–Q plots) and the Shapiro-Wilk test. In all cases, the significance threshold for deviation from normality was set at $p < 0.05$. Non-parametric statistical methods were applied for data analysis. Differences between two independent groups for quantitative variables were evaluated using the Mann–Whitney U-test. Pearson's χ^2 test was used to assess associations between qualitative variables.

RESULTS

A total of 24 samples of malignant large-intestinal neoplasms were examined. Desmin expression was identified in all analysed samples. The localisation of desmin varied: in 9 samples, desmin expression was observed exclusively in the surrounding stroma of the organ, and no expression was detected within the tumour itself. In 8 samples, by contrast, desmin was present exclusively in tumour structures, with no expression observed at the tumour periphery. In 7 samples, desmin was present both within the tumour and in the stroma. It should be noted that the amount of desmin also varied: in some cases, desmin occupied a large proportion of the section area and was well distinguishable, whereas in others it was barely visible. In samples where desmin was present exclusively in the stroma, its expression was almost always extensive (Fig. 1). However, samples with scant stromal desmin expression were also present, although they constituted a minority. In samples with desmin expression only in tumour structures, its amount varied considerably: in some cases, expression was extensive and occupied a significant part of the field of view, whereas in others desmin appeared as isolated deposits and was barely noticeable (Fig. 2). In addition, a pattern of stromal desmin expression was often observed in which the stroma appeared to "encircle" the tumour structures. It should be noted that desmin expression in areas of mucosa unaffected by tumour structures, which were present in the analysed samples, had its

own characteristics: desmin was located exclusively within the muscularis mucosae. Its amount was extremely small and appeared as a thin band. No desmin expression was observed in the crypt regions of normal mucosa. The described pattern was identical in all samples that contained areas of normal mucosa (Fig. 3).

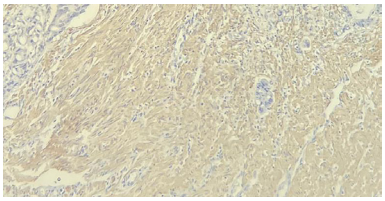

Figure 1. Extensive desmin expression in the tumour stroma
Note: immunohistochemical staining using monoclonal antibodies against desmin; magnification ×100
Source: authors' image

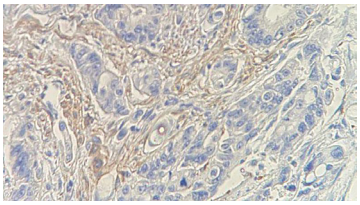

Figure 2. Weak desmin expression in tumour structures
Note: immunohistochemical staining using monoclonal antibodies against desmin; magnification ×400
Source: authors' image

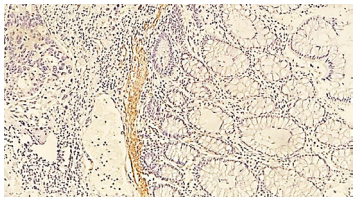

Figure 3. Desmin expression in the muscularis mucosae unaffected by the tumour
Note: Immunohistochemical staining using monoclonal antibodies against desmin; magnification ×100
Source: authors' image

The relationship between desmin expression and tumour nodule size was then examined. The area of the tumour nodules ranged from 3.8 to 70.0 square centimetres. The mean nodule area in the sample was 30.4 square centimetres. For further analysis, the material was divided into four groups: the first group included samples with a tumour nodule area of less than 15 square centimetres; the second group included cases with areas ranging from 15 to 30 square centimetres; the third group comprised tumour nodules with areas from 30 to 45 square centimetres; and the fourth group included material with tumour nodule areas of 45 square centimetres or more. Each group consisted of 7, 6, 6 and 5 samples, respectively. For quantitative assessment of desmin expression, the mean volumetric density of desmin was calculated by determining the ratio between the area occupied by desmin in a given region and the total section area. Volumetric density was calculated both for all desmin present in the sample, localised in the stroma and in tumour structures (hereafter referred to as total volumetric density), and separately for desmin located exclusively within tumour structures (hereafter referred to as volumetric density of desmin in tumour structures).

At the first stage, the relationship between desmin expressed directly within tumour structures and tumour nodule area (S) was examined. The volumetric density of desmin in tumour structures in the group with a nodule area of less than 15 square centimetres was 1.29%; in the group with an area of 15-30 square centimetres it was 3.04%; in the group with an area of 30-45 square centimetres it was 4.13%; and in the group with an area greater than 45 square centimetres it was 5.23%. These data indicated a tendency towards an increase in the amount of desmin within the tumour itself as tumour nodule size increased; however, the findings did not reach statistical significance ($p > 0.05$, Mann-Whitney test).

At the second stage, the relationship between the mean total amount of desmin (both in the tumour itself and in the stroma) and tumour nodule area was investigated. The total volumetric density of desmin in the group with a tumour nodule area of less than 15 square centimetres was 4.38%; in the group with an area of 15-30 square centimetres it was 8.08%; in the group with an area of 30-45 square centimetres it was 11.92%; and in the group with a tumour nodule area greater than 45 square centimetres it was 13.51%. These values indicated a tendency towards an increase in the total desmin content in the sample as tumour nodule size increased. Statistical analysis of the obtained results demonstrated a high level of significance ($p < 0.05$, Mann–Whitney test). For greater clarity, the above data are presented in Table 1.

Table 1. Comparison of desmin volumetric density with tumour nodule area

Tumour nodule area (S)	$S < 15$ cm^2	$15 \le S < 30$ cm^2	$30 \le S < 45$ cm^2	$S \ge 45$ cm^2
Total volumetric density of desmin, %	4.38	8.08	11.92	13.51
Volumetric density of desmin in tumour structures, %	1.29	3.04	4.13	5.23

Source: calculated by the authors

In addition, the number of cases in which desmin was present within tumour structures and cases in which no tumour desmin expression was observed was compared with tumour nodule area. In the group with a tumour nodule area of less than 15 square centimetres, there were 2 cases with desmin present in the tumour and 5 cases without such presence. In the group with a tumour nodule area greater than 45 square centimetres, there were 4 cases with desmin present in the tumour and 1 case without it. These data indicated a

tendency towards an increase in the number of samples in which desmin was present within the tumour as tumour nodule area increased; however, statistical analysis showed that the findings were not significant (p = 0.079, chi-squared test) (Table 2). This outcome might have been due to the small number of samples in the groups, particularly in the second group. It was therefore necessary to repeat the described calculations with larger sample sizes in all groups to obtain a more complete and reliable picture.

Table 2. Statistical analysis of the comparison between the presence of desmin expression within tumour structures and tumour nodule area

Four-field table analysis	S < 15 cm^2	S ≥ 45 cm^2
Number of cases with desmin present in tumour structures	2	4
Number of cases with desmin absent in tumour structures	5	1
Chi-squared test	value: 3.086	significance level: 0.079

Source: developed by the authors

The data indicating an increase in the total amount of desmin with increasing tumour nodule size were statistically significant. At the same time, there was a tendency towards an increase both in the amount of desmin located directly within tumour structures and in the number of cases in which desmin was present in the tumour as tumour nodule size increased; however, these findings did not reach statistical significance. It should be taken into account that the calculations were performed on relatively small sample groups; therefore, to clarify the results, it will be necessary in future to increase the number of samples in all groups examined and to repeat the described calculations.

DISCUSSION

The increase in the intensity of desmin expression in colorectal carcinoma samples with increasing tumour nodule size could theoretically indicate a greater potential of such tumours for further progression, invasion and metastasis. At present, the scientific literature contains a limited number of studies specifically examining the role of desmin expression in the pathogenesis and progression of colorectal cancer. In the review by G. Gomes *et al.* [15], an analysis of publication activity related to the term "desmin" was carried out. The authors noted that over recent decades the number of studies devoted to investigating its functions and its role in the pathogenesis of different diseases remained limited. It was also emphasised that a substantial proportion of publications on this topic belonged to a narrow group of authors, indicating insufficient involvement of the wider research community and highlighting the relevance of expanding investigations in this field. The overwhelming majority of existing publications concentrated on more general aspects, such as correlations between desmin levels and the invasive and metastatic potential of tumours of various localisations, as well as on its value as an immunohistochemical marker for identifying invasion into different structures. In the study by Y. Ma *et al.* [16], a systematic comparative analysis of desmin expression patterns at consecutive stages of colorectal oncogenesis was conducted: from normal colonic mucosa through the adenoma stage to primary adenocarcinoma and, finally, to its hepatic metastases. The results demonstrated a clear gradient: the level of expression of this marker consistently increased and reached its maximum in liver metastases. This upward trend correlated with clinical outcome: high desmin expression was statistically significantly associated with reduced overall patient survival. All these findings indirectly suggested a high progression potential of tumours with pronounced desmin expression, which could correlate with the results obtained in the present study. In the research by J. Shin *et al.* [17], the diagnostic and prognostic value of desmin as a marker of venous invasion was assessed. Through the use of dual immunohistochemical staining (CD31/Desmin), the authors demonstrated that morphologically verified venous invasion served as an independent factor associated with a statistically significant reduction in survival among patients with pancreatobiliary malignancies. The problem of objectively verifying early invasion in colorectal cancer which directly influences therapeutic decision-making was further developed in the study by M. Yoshida *et al.* [11]. That work systematically examined and confirmed the clinico-morphological value of desmin as an immunohistochemical marker for assessing the condition of the muscularis mucosae. The use of this method ensured reproducible differentiation between pseudoinvasion and true invasion and allowed accurate quantitative measurement of carcinoma penetration into the submucosa, which was of primary importance for correct staging and prognostication. Thus, an analysis of contemporary studies revealed the formation of a stable paradigm in which the assessment of desmin expression was regarded as a promising method for predicting tumour progression. The empirical data obtained in the present study were consistent with this emerging trend, mutually complementing and strengthening the evidence base for the clinical application of this marker. The work of Q. Yan *et al.* [18] highlighted the importance of distinguishing macroscopic tumour size as a separate, significant prognostic category. Their analysis convincingly demonstrated that a critical diameter of 5 cm served as a clear marker of increased biological

aggressiveness, directly correlating with poorer prognosis. This observation allowed tumour diameter to be viewed not merely as a descriptive characteristic but as an independent predictor deserving consideration in risk stratification. Within the framework of analysing factors determining tumour-cell dissemination, H. Feng *et al.* [19] empirically confirmed a significant positive correlation between the macroscopic size of the primary lesion and its metastatic capacity. This finding allowed tumour size to be considered not only as a staging parameter but also as an indirect indicator of its invasive and disseminative potential. Of particular interest were the results of the study by T. Kato *et al.* [20], which examined the relationship between characteristics of the primary tumour and the risk of developing metachronous colorectal cancer. The authors established that primary tumour size served as a strong independent predictor of this risk. Notably, the prognostic sensitivity of this parameter for metachronous cancer was more than twice its significance in the context of synchronous tumours, underscoring the fundamentally different prognostic roles of tumour size in these two clinical scenarios. In the work of W. Dai *et al.* [21], the relationship between the macroscopic characteristic of tumour size and long-term oncological outcomes in colorectal cancer was systematically investigated. The data convincingly showed that the volume of the primary lesion constituted a significant factor integrating biological properties such as duration of growth and invasive potential, which ultimately manifested in reduced survival among patients with larger neoplasms. Based on this, close examination of tumour progression characteristics particularly the analysis of expression patterns of specific proteins was of great importance. The data presented in the present study indicated the presence of a direct relationship between the amount of expressed desmin and tumour size, suggesting its potential prognostic significance. However, further research is required to draw definitive conclusions regarding the clinical value of desmin as a marker, including detailed comparison of the presence and intensity of its expression with long-term survival in patient cohorts.

Based on the results of the present study, it could be concluded that desmin was a promising biomarker involved in key aspects of colorectal cancer progression. The established positive correlation between its expression level and the size of the primary tumour was consistent with the emerging scientific paradigm in which desmin was associated with invasive growth, metastasis and an unfavourable prognosis. However, this field remained insufficiently developed, as evidenced by the scarcity of large targeted studies. This defined the main directions for further research. A promising avenue involved conducting large-scale prospective studies aimed at formalising the clinico-morphological significance of desmin. The key objectives would include: 1) standardising methods for quantitative assessment of desmin expression (immunohistochemical scoring, digital image analysis); 2) determining relevant diagnostic threshold values enabling risk stratification; 3) validating the independent prognostic value of desmin in multivariate models incorporating traditional parameters (stage, size, grade). The implementation of these measures could make it possible to move from a fundamental understanding of the role of desmin to its practical use as a diagnostic and prognostic marker.

CONCLUSIONS

Desmin expression was identified in all 24 analysed samples. Desmin was expressed both in the stroma distant from tumour structures and within the tumour itself. In the stroma, desmin was more frequently expressed extensively and appeared as layers or thick bands. In the vicinity of the tumour, the amount of desmin varied: it was visualised as isolated deposits, thin bands, or occupying a substantial portion of the section. In areas of mucosa unaffected by tumour structures, desmin in all examined cases was located exclusively within the muscularis mucosae, where it appeared as a thin band. The conducted study made it possible to characterise in detail the patterns of desmin expression in colorectal cancer tissue and to establish their relationship with primary tumour size. A key finding of the work was the identification of a statistically significant ($p < 0.05$) positive correlation between total desmin volumetric density and tumour nodule area. The total volumetric density of desmin in the group with a tumour nodule area of less than 15 square centimetres was 4.38%; in the group with an area of 15-30 square centimetres it was 8.08%; in the group with an area of 30-45 square centimetres it was 11.92%; and in the group with an area greater than 45 square centimetres it was 13.51%. These findings indicated that as tumour mass increased, a systemic rise in desmin expression occurred, potentially reflecting activation of stromal processes and mesenchymal transformation associated with disease progression. An evident, though not statistically significant ($p > 0.05$), tendency was also observed towards an increase both in the volumetric density of desmin within tumour structures and in the frequency of its presence in the tumour in groups with larger neoplasms. Thus, the obtained results were consistent with the hypothesis that desmin was involved in tumour progression processes in colorectal cancer and supported its potential role as a marker associated with biological aggressiveness. To confirm these findings definitively particularly regarding desmin expression within tumour cells and to establish accurate prognostic threshold values, further studies involving larger and more representative patient cohorts are required.

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CONFLICT OF INTEREST

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Колоректалдык карциномалардагы десмин экспрессиясынын шишик көлөмү менен байланышы

Андрей Филин

Медицина илимдеринин кандидаты, доцент
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси
«Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0000-0003-1670-3694

Елена Припутневич

Ординатор
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси
«Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0000-0002-7763-9932

Виктория Шишкина

Медицина илимдеринин кандидаты, доцент
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси
«Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0000-0001-9185-4578

Аннотация. Булчуң клеткалары, миофибробласттар жана айрым патологиялык клеткалар тарабынан экспрессияланууучу десмин дүйнөдө кездешүү жыштыгы боюнча үчүнчү орунда турган колоректалдык рактын морфогенези контекстинде караганда азыркыга чейин жетишсиз изилденген. Изилдөөнүн максаты колоректалдык карциномаларда десминдин экспрессиясынын өзгөчөлүктөрүн аныктоо жана экспрессиянын деңгээлин шишик түйүнүнүн көлөмү менен салыштыруу болуп саналат. Изилдөөнүн жүрүшүндө жоон ичегиден алынган 24 даана залалдуу шишик үлгүсү гистологиялык жактан изилденип, иммуногистохимиялык ыкмасы аркылуу десминге каршы моноклоналдык чычкан антителолору колдонулду. Десминдин экспрессиясы бардык изилденген үлгүлөрдөн аныкталды. Десмин шишик түзүмдөрүндө да, органды курчап турган стромада да экспрессияланды. Ар кандай үлгүлөрдө десмин экспрессиясынын сандык гетерогендүүлүгү белгиленип, ал жогорку (массивдүү) деңгээлден төмөнкү деңгээлге чейин өзгөрүп турганы аныкталды. Десминдин көлөмдүк тыгыздыгын шишик түйүнүнүн аянты менен салыштырганда, жалпы экспрессияланган десминдин көлөмү ири шишик түйүндөрү бар үлгүлөрдө статистикалык жактан ишенимдүү түрдө көбүрөөк экени белгиленди. Мындан тышкары, шишик аянты чоңойгон сайын шишик түзүмдөрүндө локализацияланган десминдин көлөмү өсүү тенденциясы байкалды. Ошондой эле шишикте десминдин болуш-болбош фактысы шишик конгломератынын өлчөмү менен салыштырылды. Натыйжада ири шишик түйүндөрүндө десминдин шишик ичинде кездешүү жыштыгы кичине өлчөмдөгү шишиктерге салыштырмалуу кыйла жогору экени аныкталды. Алынган жыйынтыктар десминди шишик прогрессиясынын тобокелдигин баалоодо жана потенциалдуу прогностикалык маркер катары колдонуу мүмкүндүгүн көрсөтөт

Негизги сөздөр: шишиктин көлөмү; аралык филаменттердин белогу; строма; иммуногистохимиялык ыкма; шишик түйүнү

Связь экспрессии десмина с размером опухоли в колоректальных карциномах

Андрей Филин
Кандидат медицинских наук, доцент
Федеральное государственное бюджетное образовательное учреждение высшего образования
«Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0000-0003-1670-3694

Елена Припутневич
Ординатор
Федеральное государственное бюджетное образовательное учреждение высшего образования
«Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0000-0002-7763-9932

Виктория Шишкина
Кандидат медицинских наук, доцент
Федеральное государственное бюджетное образовательное учреждение высшего образования
«Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0000-0001-9185-4578

Аннотация. Десмин, экспрессируемый мышечными клетками, миофибробластами и некоторыми патологическими клетками, остаётся слабо изученным в контексте морфогенеза колоректального рака – третьего по распространённости онкологического заболевания в мире. Целью работы явилось изучение особенностей экспрессии десмина в колоректальных карциномах, а также сопоставление выраженности экспрессии с размером опухолевого узла. В ходе работы гистологическому исследованию были подвергнуты 24 образца злокачественных опухолей толстого кишечника, окрашенных иммуногистохимическим методом с применением моноклональных мышиных антител к десмину. Экспрессия десмина была выявлена во всех исследуемых образцах. Десмин экспрессировался как в опухолевых структурах, так и в окружающей строме органа. Выявлена количественная неоднородность экспрессии десмина, варьирующая от массивной до скудной в различных образцах. При сопоставлении объемной плотности десмина с площадью опухолевого узла было выявлено, что общее количество экспрессированного десмина было статистически значимо большим в образцах с крупными опухолевыми узлами. Кроме того, была обнаружена тенденция к увеличению количества десмина, локализованного в опухолевых структурах с возрастанием площади опухоли. В ходе работы также проводилось сопоставление факта наличия десмина в опухоли с величиной опухолевого конгломерата. Выяснилось, что в крупных опухолевых узлах десмин присутствовал в опухоли значительно чаще, чем в образцах с небольшими по размеру опухолями. Полученные в ходе исследования результаты говорят о потенциальной возможности использования десмина для оценки риска прогрессии опухоли, а также потенциального его использования как прогностического маркера

Ключевые слова: объем опухоли; белок промежуточных филаментов; строма; иммуногистохимический метод; опухолевый узел