

# Identification of microRNA signatures in thyroid cancer

A. Ya. Pasko<sup>id</sup> A,B,C,D, V. D. Skrypko<sup>id</sup> C,E,F

Ivano-Frankivsk National Medical University, Ukraine

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of the article

## Keywords:

рак щитоподібної залози, папілярна карцинома, фолікулярна карцинома, мікроРНК, біомаркери.

## Ключові слова:

thyroid cancer, papillary carcinoma, follicular carcinoma, microRNA, biomarkers.

Надійшла до редакції /  
Received: 22.01.2026

Після доопрацювання /  
Revised: 12.03.2026

Схвалено до друку /  
Accepted: 20.03.2026

Конфлікт інтересів:  
відсутній.

Conflicts of interest:  
authors have no conflict  
of interest to declare.

**Aim:** to identify differentially expressed microRNAs (miRNAs) in papillary and follicular thyroid cancer (TC) and to evaluate their potential as diagnostic and prognostic biomarkers.

**Materials and methods.** Three GEO datasets were analyzed: GSE104006 (20 samples of papillary TC and 6 normal tissues), GSE191117 (50 samples of papillary TC and 50 normal samples), and GSE62054 (17 samples of follicular carcinoma and 8 benign tumors). Differentially expressed miRNAs were identified with  $|\log_2FC| > 1$  and  $p < 0.05$  with subsequent FDR correction DIANA miRPath v. 3 was used to assess signaling pathways, miRNet v. 2.0 to identify target genes, and ShinyGO v. 0.82 for functional annotation. Prognostic significance was evaluated using ENCORI database.

**Results.** Six miRNAs were selected: *hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484*. They potentially regulate over 2,000 target genes, including *RET*, *CCND1*, *TP53*, *HIF1A*, *IL6*, and *IL1B*, which are associated with the development and progression of malignant tumors. GEO analysis revealed their involvement in the regulation of metabolism, biosynthesis, protein modification, as well as in the binding functions of transcription factors, DNA, and RNA. Nineteen KEGG signaling pathways were identified, 13 of which are closely associated with carcinogenesis. Prognostic analysis indicated that low expression of *hsa-miR-146b-5p* and *hsa-miR-221-5p* correlate with significantly poorer overall survival in TC patients.

**Conclusions.** The proposed panel of six miRNAs may have significant potential for the differential diagnosis of papillary and follicular thyroid cancer, risk stratification, and prognosis; *hsa-miR-146b-5p* and *hsa-miR-221-5p* demonstrated the greatest prognostic value. Further experimental studies are needed for the clinical validation of these biomarkers.

**Modern medical technology. 2026;18(2):105-111**

## Ідентифікація сигнатур мікроРНК при раку щитоподібної залози

А. Я. Пасько, В. Д. Скрипко

**Мета роботи** – визначити диференційно експресовані мікроРНК (міРНК) при папілярному та фолікулярному раку щитоподібної залози (РЩЗ) та оцінити їх потенціал як діагностичних і прогностичних біомаркерів.

**Матеріали і методи.** Для аналізу використано три бази даних GEO: GSE104006 (20 зразків папілярного РЩЗ і 6 нормальних тканин), GSE191117 (50 зразків папілярного РЩЗ і 50 нормальних тканин), GSE62054 (17 зразків фолікулярного раку та 8 доброякісних новоутворень). Диференційно експресовані міРНК визначали за критеріями  $|\log_2FC| > 1$ ,  $p < 0,05$ , із подальшою корекцією FDR. Для оцінювання сигнальних шляхів використано DIANA miRPath v. 3, для виявлення генів-мішеней – miRNet v. 2.0, для функціонального аналізу – ShinyGO v. 0.82. Прогностичну значущість перевіряли за допомогою бази ENCORI.

**Результати.** Встановлено панель із шести ключових міРНК: *hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, *hsa-miR-484*. Вони потенційно регулюють понад 2000 генів, серед них – *RET*, *CCND1*, *TP53*, *HIF1A*, *IL6* та *IL1B*, що асоційовані з розвитком і прогресуванням злоякісних пухлин. Аналіз GEO дав змогу визначити їхню участь у регуляції метаболізму, біосинтезу, модифікацій білків, а також у функціях зв'язування транскрипційних факторів, ДНК і РНК. Ідентифіковано 19 сигнальних шляхів, із них 13 тісно пов'язані з канцерогенезом. Прогностичний аналіз показав, що низькі рівні експресії *hsa-miR-146b-5p* та *hsa-miR-221-5p* корелюють із гіршою загальною виживаністю.

**Висновки.** Запропоновано панель із шести міРНК, що може мати високий потенціал для диференційної діагностики папілярного й фолікулярного РЩЗ, стратифікації ризику та прогнозування перебігу захворювання. Найбільше прогностичне значення мали *hsa-miR-146b-5p* і *hsa-miR-221-5p*. Доцільно продовжити експериментальні дослідження для клінічної валідації цих біомаркерів.

**Сучасні медичні технології. 2026. Т. 18, № 2(69). С. 105-111**

© The Author(s) 2026  
This is an open access article  
under the  
[Creative Commons](#)  
[CC BY-NC 4.0 license](#)

Thyroid cancer (TC) is the most common malignant neoplasm of the endocrine system, accounting for more than 90 % of all malignancies of this localization [1]. Over the past few decades, there has been a steady global increase in TC incidence, which can be attributed to both improvements in diagnostic methods (particularly ultrasound examination and fine-needle aspiration biopsy) and a real rise in tumor detection rates associated with environmental factors and lifestyle changes. Within the overall structure of cancer morbidity, TC occupies a leading position among women of young and middle age [2].

Histologically, TC comprises several subtypes that differ in clinical course, prognosis, and therapeutic responsiveness. The most frequent form is papillary thyroid carcinoma, representing 75–85 % of all cases, which is characterized by a relatively favorable prognosis and high sensitivity to radioactive iodine therapy. Follicular carcinoma accounts for 10–15 % of cases and is distinguished by a high risk of hematogenous metastasis, contributing to a less favorable disease course. Medullary carcinoma, arising from parafollicular C-cells, is a less common condition (approximately 5 % of cases) and is frequently associated with *RET* gene mutations, including those found in hereditary multiple endocrine neoplasia syndromes. The most aggressive form is anaplastic thyroid carcinoma, diagnosed in less than 2 % of cases, which is marked by rapid invasive growth and an extremely poor prognosis [3].

Clinically, early-stage TC is often asymptomatic and typically detected only through ultrasound screening [4]. Diagnostic workup includes a comprehensive approach, including ultrasound with elastography, scintigraphy, and cytological examination of material obtained via fine-needle aspiration biopsy.

In recent years, increasing attention has been focused on molecular markers that may improve the accuracy of diagnosis, prognosis, and therapy personalization in TC. One of the most promising research areas is the study of microRNAs (miRNAs) – small non-coding RNAs that regulate gene expression at the post-transcriptional level [5]. Dysregulation of miRNAs in tumor cells and at the systemic level has been linked to the activation of oncogenic pathways, inhibition of tumor suppressor genes, and remodeling of the tumor microenvironment. Expression profiling may thus enhance differential diagnosis, as well as prediction of disease progression and treatment response [6].

Taken together, TC represents a heterogeneous group of neoplasms with diverse clinical behavior and outcomes. Despite generally high survival rates, the timely differential diagnosis, identification of aggressive forms, and development of novel therapeutic strategies remain pressing issues [7]. Research into miRNAs opens new perspectives for the development of biomarker panels that may serve not only as diagnostic and risk stratification tools but also as potential therapeutic targets [8].

## Aim

The aim of this study was to identify differentially expressed microRNAs in papillary and follicular thyroid cancer and evaluate their potential as diagnostic and prognostic biomarkers.

## Material and methods

The study was based on datasets obtained from the Gene Expression Omnibus (GEO) of the [National Center for Biotechnology Information](#), which provides information on microRNA expression in normal, benign, and malignant thyroid tissues. Specifically, dataset [GSE104006](#) was used, comprising microRNA expression profiles from non-transformed thyroid tissue (n = 6) and papillary thyroid carcinoma (PTC) tissue (n = 20). Dataset [GSE191117](#) provided information on microRNA expression in 50 normal thyroid tissue samples and 50 PTC samples. Additionally, dataset [GSE62054](#) was employed to evaluate microRNA expression in benign thyroid neoplasms (n = 8) and follicular thyroid carcinoma (FTC) tissue (n = 17).

MicroRNA expression profiles from the selected datasets were analyzed using the online tool [GEO2R](#). For datasets GSE104006 and GSE191117, microRNAs with  $|\log_2$  fold change (FC)| >1 and p-value <0.05 were considered differentially expressed between PTC and normal thyroid tissue. For dataset GSE62054, differentially expressed microRNAs between FTC and benign thyroid tissue were identified using  $|\log_2$  fold change (FC)| >0 as the threshold. To reduce the risk of false-positive results, adjusted p-values (adj.p) were calculated using the Benjamini & Hochberg false discovery rate (FDR) method. Differences were considered statistically significant at adj.p < 0.05. The results obtained were visualized as volcano plots.

Pathway enrichment analysis of the identified microRNAs was performed using [DIANA miRPath v. 3.0](#) with the following parameters: KEGG pathway database, Fisher's exact test, TarBase v. 7.0 as the reference, significance threshold p < 0.05, and FDR correction for multiple testing. Hierarchical clustering and heatmap visualization of microRNAs and KEGG signaling pathways were generated using DIANA Tools miRPath v. 3.

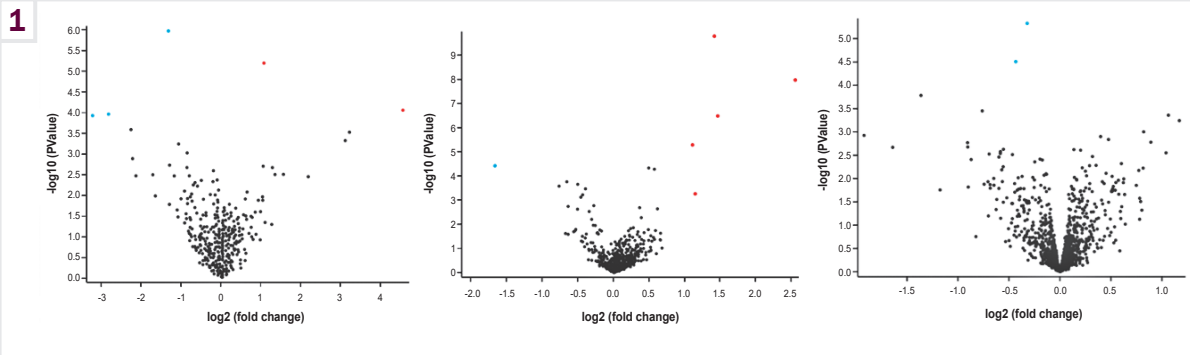
Target gene identification of the selected microRNAs was carried out using miRNet v. 2.0, based on the [TarBase v7.0 database](#), which contains experimentally validated microRNA-target interactions across 18 species, including *Homo sapiens*.

Pathway analysis of the identified target genes was performed using [ShinyGO v. 0.82](#). Gene Ontology (GO) analysis included three categories: molecular functions, biological processes, and cellular components. A significance threshold of p < 0.05 was applied. The classification results by biological processes and molecular functions were visualized as dot plots.

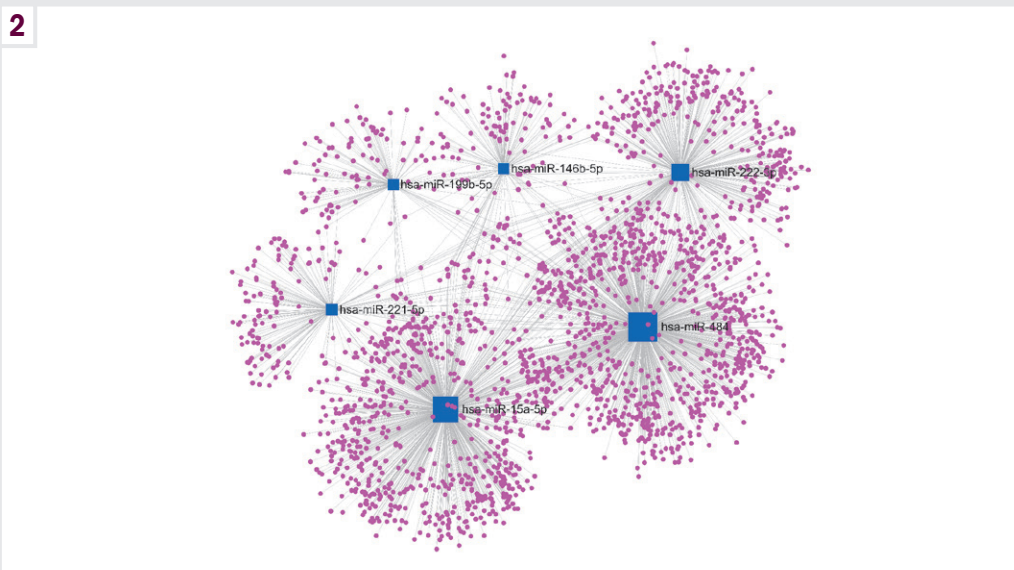
Finally, survival analysis based on the expression levels of the identified microRNA panel was performed using [ENCORI](#). Along with gene expression data from 32 cancer types, ENCORI enables pan-cancer analysis of RNA–RNA and RNA–RBP interactions and provides integrated platforms for survival and differential expression analysis of microRNAs, lncRNAs, pseudogenes, and mRNAs.

## Results

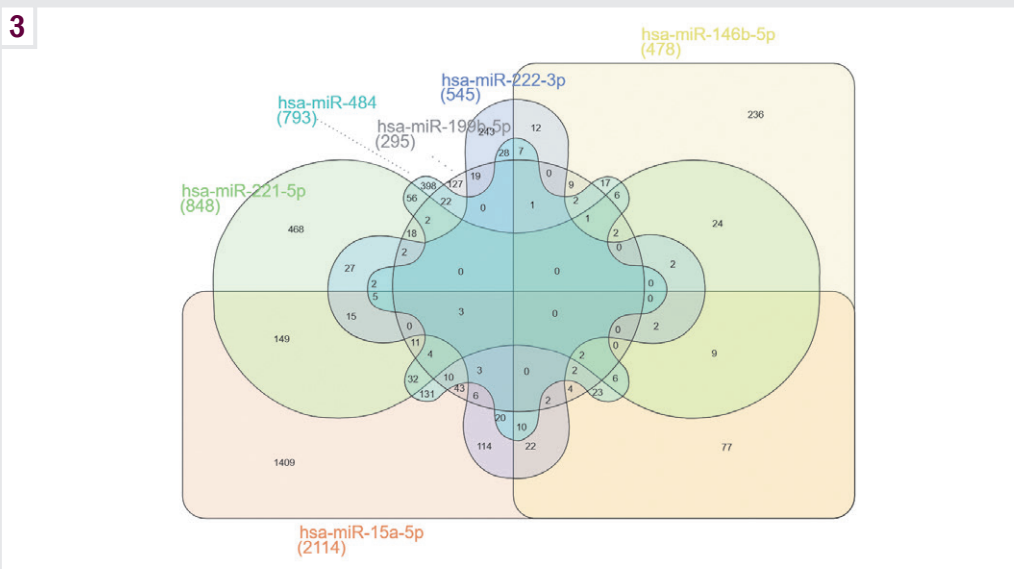
Analysis of the GSE104006 dataset identified five differentially expressed microRNAs (DEMs) in malignant papillary thyroid carcinoma (PTC) tissue compared with non-transformed thyroid tissue: *hsa-miR-100-5p*, *hsa-miR-199b-5p*, *hsa-miR-146b-5p*, *hsa-miR-7-5p*, and *hsa-miR-15a-5p*. Examination of



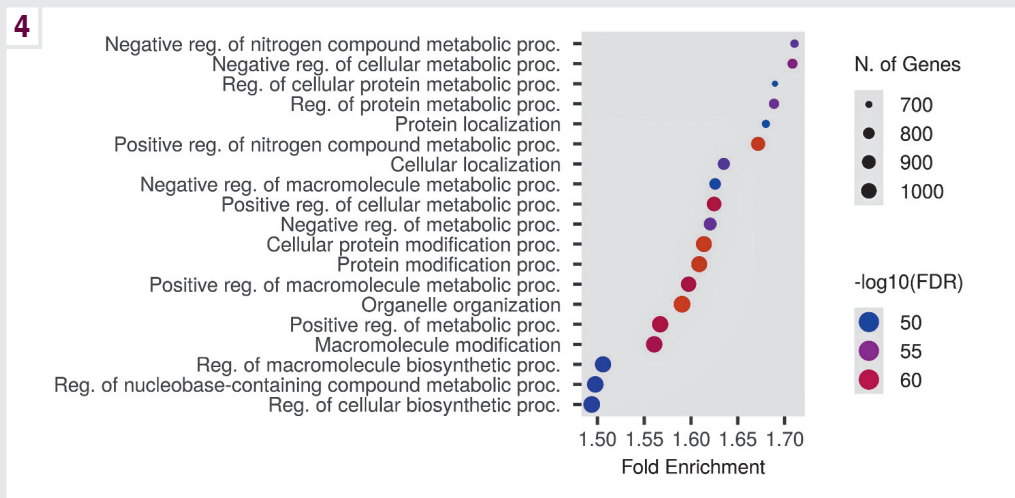
**Fig. 1.** Expression profile of tumor-associated miRNAs in patients with thyroid cancer (TC). Volcano plots representing DEMs identified in datasets GSE104006 (A), GSE191117 (B), and GSE62054 (C). Volcano plots display both p-values and fold changes in miRNA expression. Blue and red dots represent miRNAs with lower and higher expression levels, respectively, relative to the comparison group.



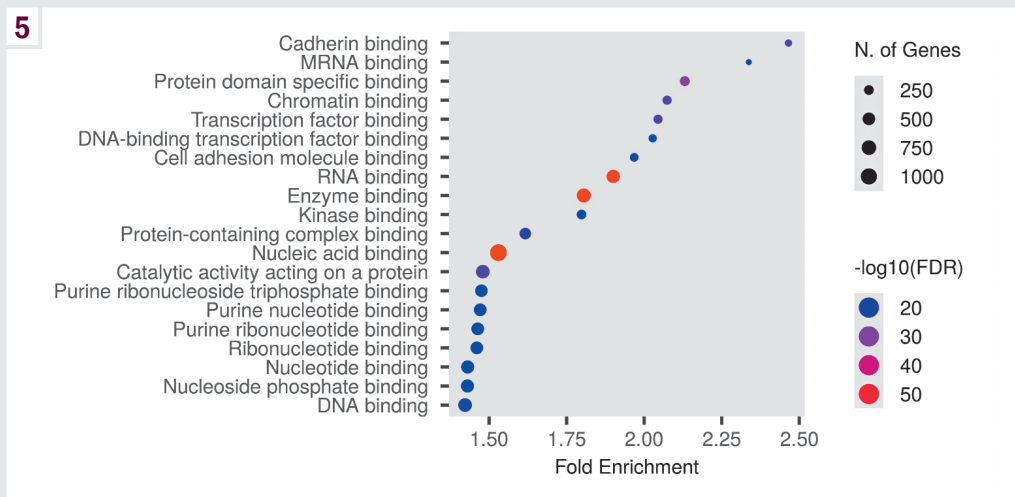
**Fig. 2.** Schematic representation of the regulatory network of the studied miRNAs. Squares indicate miRNAs, while circles represent their target genes.



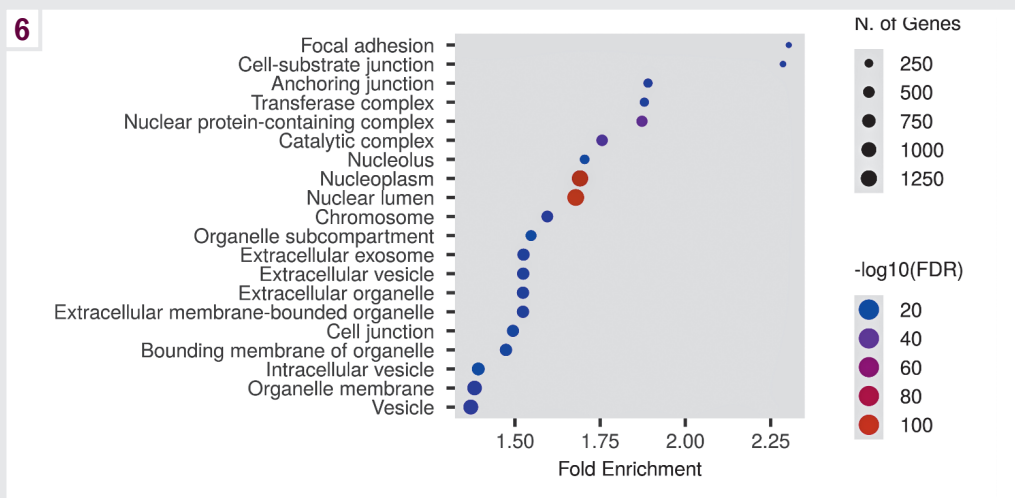
**Fig. 3.** Venn diagram illustrating the overlap of target gene sets regulated by the studied miRNAs.



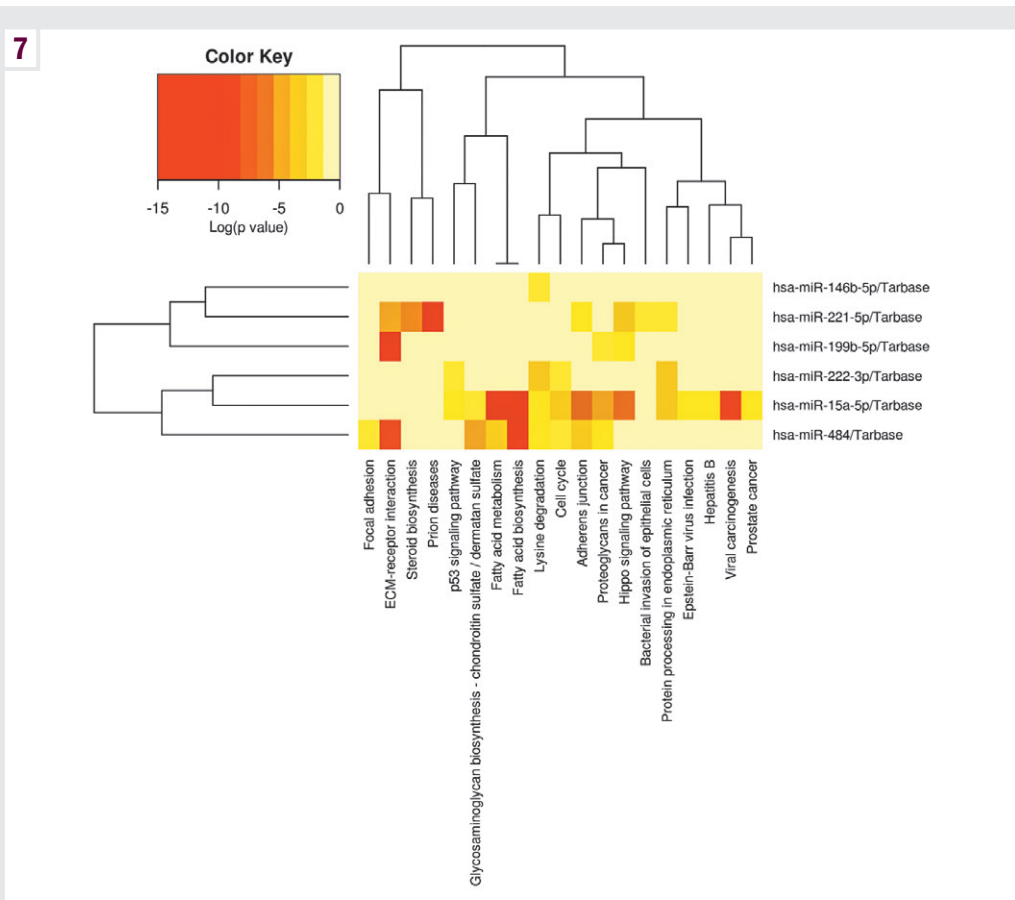
**Fig. 4.** Biological processes involving protein products of target genes regulated by *hsa-miR-15a-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484*.



**Fig. 5.** Molecular functions of proteins encoded by target genes regulated by *hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484*.



**Fig. 6.** Localization of protein products of target genes of *hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484* within cellular components.



**Fig. 7.** Hierarchical clustering and heatmap of miRNA / KEGG signalling pathways regulated by the studied miRNAs.

the GSE191117 dataset, which contains microRNA expression profiles in PTC tissue, revealed six dysregulated microRNAs associated with the development of this malignancy: *hsa-miR-222-3p*, *hsa-miR-146b-5p*, *hsa-miR-221-3p*, *hsa-miR-221-5p*, *hsa-miR-15a-5p*, and *hsa-miR-7-5p*.

In the GSE62054 dataset, two microRNAs were identified as dysregulated in follicular thyroid carcinoma (FTC): *hsa-miR-607* and *hsa-miR-484*. Notably, no microRNA was consistently dysregulated across all three analyzed datasets. However, altered expression of three miRNAs (*hsa-miR-146b-5p*, *hsa-miR-7-5p*, and *hsa-miR-15a-5p*) was observed in both GSE104006 and GSE191117 (Fig. 1).

Based on these findings and supporting evidence from the literature, we selected a panel of six miRNAs for further analysis: *hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484*.

According to TarBase v. 7.0, *hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484* may regulate the expression of 2,170 genes. Analysis of these target genes using DisGeNET v. 7.0 and the Hypergeometric test identified seven genes (*CCND1*, *RET*, *TP53*, *HIF1A*, *BRD4*, *IL6*, and *IL1B*) whose dysregulation is associated with the onset and progression of thyroid malignancies ( $p = 0.0015$ ). In addition, five genes (*CCND1*, *CDKN1A*, *OCRL*, *RET*, and *CDKN1B*) regulated by the selected miRNAs were

found to be linked to the development of benign thyroid neoplasms ( $p = 0.0000677$ ) (Fig. 2).

A detailed analysis of target genes revealed that *hsa-miR-15a-5p* regulates 2,114 genes, *hsa-miR-146b-5p* – 478 genes, *hsa-miR-199b-5p* – 295 genes, *hsa-miR-221-5p* – 848 genes, *hsa-miR-222-3p* – 545 genes, and *hsa-miR-484* – 793 genes (Fig. 3).

To determine the functional significance of the analyzed miRNAs, a GO analysis of their target genes was performed based on the information from TarBase v. 7.0, using the online tool ShinyGO v. 0.82. In particular, three main ontological categories were analyzed: “biological processes”, “cellular components”, and “molecular functions”, which are presented in the form of chord diagrams.

It was established that among the biological processes involving the target genes of the studied miRNAs, the most prevalent are the regulation of metabolic processes, protein modifications, and biosynthesis (Fig. 4).

It was found that the protein products of the genes regulated by the studied miRNAs are involved in such molecular functions as cadherin binding, mRNA binding, chromatin binding, transcription factor binding, enzyme binding, DNA binding, RNA binding, and others (Fig. 5).

It was determined that the protein products of genes regulated by the identified miRNAs are most frequently localized in organelle membranes, intracellular vesicles, and extracellular vesicles (Fig. 6).

At the next stage, signaling pathway analysis regulated by the identified panel of six miRNAs was performed using DIANA Tools miRPath v. 3 (accessed May 20, 2025) and the TarBase v. 7.0 database. The results are presented as hierarchical clusters and a heatmap of miRNA/KEGG signaling pathways, reflecting the most relevant pathways regulated by these miRNAs. As shown in Fig. 7, this miRNA group is associated with a total of 19 signaling pathways, with strong associations observed for 13 pathways.

To assess the prognostic significance of the identified miRNA panel, bioinformatic analysis of the association between their expression levels and survival outcomes in patients with thyroid cancer was performed using the ENCORI database. Patients with low expression levels of *hsa-miR-146b-5p* and *hsa-miR-221-5p* in thyroid carcinoma tissue exhibited significantly lower survival compared to patients whose tumor tissue showed higher levels of these miRNAs. No significant differences in overall survival were observed according to the expression levels of *hsa-miR-15a-5p*, *hsa-miR-199b-5p*, *hsa-miR-222-3p*, or *hsa-miR-484* in thyroid cancer tissue.

## Discussion

The present study provides a comprehensive bioinformatic characterization of miRNA deregulation in papillary and follicular thyroid cancer, integrating data from multiple independent GEO datasets. By combining differential expression analysis, target gene prediction, functional annotation, pathway enrichment, and survival analysis, we identified a six-miRNA panel that appears to play a central role in thyroid carcinogenesis and tumor progression.

Although no single miRNA was consistently dysregulated across all analyzed datasets, the recurrent involvement of *hsa-miR-146b-5p*, *hsa-miR-15a-5p*, and *hsa-miR-7-5p* in papillary thyroid carcinoma datasets highlights the biological heterogeneity of thyroid tumors and reflects differences in histological subtype, sample composition, and experimental platforms. Such variability has been widely reported in transcriptomic studies and underscores the importance of integrative approaches that rely not only on overlap, but also on functional relevance and literature support.

The selected panel of six miRNAs (*hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484*) was shown to potentially regulate more than 2,000 target genes, many of which are key drivers of thyroid tumorigenesis. Notably, the identification of *RET*, *CCND1*, *TP53*, *HIF1A*, *IL6*, and *IL1B* among the target genes supports the biological plausibility of the findings, as these genes are critically involved in cell cycle regulation, hypoxia response, inflammation, and oncogenic signaling pathways known to be altered in thyroid cancer.

Functional enrichment analysis further demonstrated that the target genes of the identified miRNAs are predominantly involved in metabolic regulation, biosynthetic processes, and protein modification. These findings are consistent with the concept that metabolic reprogramming and altered protein turnover are hallmarks of cancer progression. Moreover, the enrichment of molecular functions related to transcription factor binding,

chromatin binding, and RNA binding suggests that the studied miRNAs exert broad regulatory control over gene expression networks rather than acting through isolated signaling cascades.

Pathway analysis revealed that the six-miRNA panel is associated with 19 KEGG signaling pathways, 13 of which are directly linked to oncogenic processes. This extensive pathway involvement indicates that these miRNAs may function as key network regulators, coordinating multiple cancer-related pathways simultaneously. Such multi-pathway regulation is particularly relevant for thyroid cancer, which is characterized by complex molecular alterations rather than a single dominant driver in many cases.

Of special interest is the prognostic significance of *hsa-miR-146b-5p* and *hsa-miR-221-5p*. Survival analysis demonstrated that low expression levels of these miRNAs are associated with significantly poorer overall survival in thyroid cancer patients. These results align with previous reports describing the role of miR-146b and miR-221 family members in tumor invasiveness, metastatic potential, and modulation of inflammatory and MAPK-related signaling pathways. Moreover, literature data indicate that these microRNAs are involved in the development, increased invasiveness, and metastasis of thyroid cancer cells [8, 9, 10, 11, 12]. The observed association with patient survival suggests that these miRNAs may serve not only as diagnostic markers but also as clinically relevant prognostic indicators.

Despite the strengths of this study, several limitations should be acknowledged. First, the analysis is based on retrospective public datasets, which may introduce bias related to sample selection and technical variability. Second, the predicted miRNA–target interactions, although experimentally validated in databases, were not confirmed in thyroid cancer – specific experimental models. Therefore, further *in vitro* and *in vivo* studies using clinical samples are required to validate the functional roles of these miRNAs and to assess their utility in clinical practice.

In summary, this study expands current knowledge on miRNA-mediated regulatory networks in thyroid cancer and highlights a panel of six miRNAs with potential diagnostic and prognostic relevance. The findings provide a solid foundation for future experimental validation and support the development of miRNA-based strategies for improved risk stratification and personalized management of thyroid cancer patients.

## Conclusions

1. A six-miRNA panel (*hsa-miR-15a-5p*, *hsa-miR-146b-5p*, *hsa-miR-199b-5p*, *hsa-miR-221-5p*, *hsa-miR-222-3p*, and *hsa-miR-484*) was identified as a potentially important regulatory component in thyroid cancer, reflecting the molecular heterogeneity of the disease.
2. GO functional analysis indicated that the target genes of the studied microRNAs are involved in the coordinated regulation of multiple cancer-related pathways, supporting their role as network-level modulators rather than a single dominant driver in thyroid carcinogenesis/tumor-related processes.
3. Among the identified miRNAs, *hsa-miR-146b-5p* and *hsa-miR-221-5p* demonstrate potential prognostic relevance as their expression levels are associated with overall survival and may serve as biomarkers for patient stratification.

**Prospects for further research:** Further experimental and clinical studies are required to validate the functional roles, target interactions, and clinical applicability of the identified microRNAs as diagnostic and prognostic biomarkers in thyroid cancer.

### Funding

This work was conducted under a scientific and technical cooperation agreement with LLC "Clinic of Personalized Diagnostic and Therapeutic Design "Onkoternostyka", Kyiv, Ukraine.

### Information about the authors:

Pasko A. Ya., MD, PhD, Associate Professor of the Department of Surgical Diseases, Ivano-Frankivsk National Medical University, Ukraine.

ORCID ID: [0000-0002-6688-7666](https://orcid.org/0000-0002-6688-7666)

Skrypko V. D., MD, PhD, DSc, Professor of the Department of Postgraduate Surgery and Urology, Ivano-Frankivsk National Medical University, Ukraine.

ORCID ID: [0000-0002-1555-2030](https://orcid.org/0000-0002-1555-2030)

### Відомості про авторів:

Пасько А. Я., канд. мед. наук, доцент каф. хірургічних хвороб, Івано-Франківський національний медичний університет, Україна.

Скрипко В. Д., д-р мед. наук, професор каф. хірургії післядипломної освіти та урології, Івано-Франківський національний медичний університет, Україна.



Андрій Пасько (Andrii Pasko)  
[apasko@ifnmu.edu.ua](mailto:apasko@ifnmu.edu.ua)

### References

1. Kitahara CM, Schneider AB. Epidemiology of thyroid cancer. *Cancer Epidemiol Biomarkers Prev.* 2022;31(7):1284-97. doi: [10.1158/1055-9965.EPI-21-1440](https://doi.org/10.1158/1055-9965.EPI-21-1440)
2. The International Agency for Research on Cancer (IARC). Global Cancer Observatory [Internet]. Who.int. [cited 2026 Jan 14]. Available from: <https://gco.iarc.who.int/factsheets/cancers/32-thyroid>
3. Asa SL. The Current Histologic Classification of Thyroid Cancer. *Endocrinol Metab Clin North Am.* 2019;48(1):1-22. doi: [10.1016/j.ecl.2018.10.001](https://doi.org/10.1016/j.ecl.2018.10.001)
4. Forma A, Klodnicka K, Pająk W, Flieger J, Teresińska B, Januszewski J, et al. Thyroid Cancer: Epidemiology, Classification, Risk Factors, Diagnostic and Prognostic Markers, and Current Treatment Strategies. *Int J Mol Sci.* 2025;26(11):5173. doi: [10.3390/ijms26115173](https://doi.org/10.3390/ijms26115173)
5. O'Brien J, Hayder H, Zayed Y, Peng C. Overview of MicroRNA Biogenesis, Mechanisms of Actions, and Circulation. *Front Endocrinol (Lausanne).* 2018;9:402. doi: [10.3389/fendo.2018.00402](https://doi.org/10.3389/fendo.2018.00402)
6. Prete A, Borges de Souza P, Censi S, Muzza M, Nucci N, Sponziello M. Update on Fundamental Mechanisms of Thyroid Cancer. *Front Endocrinol (Lausanne).* 2020;11:102. doi: [10.3389/fendo.2020.00102](https://doi.org/10.3389/fendo.2020.00102)
7. Silaghi H, Pop LA, Georgescu CE, Muntean D, Crișan D, Silaghi P, et al. MicroRNA Expression Profiling-Potential Molecular Discrimination of Papillary Thyroid Carcinoma Subtypes. *Biomedicines.* 2024;12(1):136. doi: [10.3390/biomedicines12010136](https://doi.org/10.3390/biomedicines12010136)
8. Pasko AY, Skrypko VD. [MicroRNAs as prognostic markers of thyroid carcinoma]. *Clinical Endocrinology and Endocrine Surgery.* 2025;2(90):48-52. Ukrainian. doi: [10.30978/CEES-2025-2-48](https://doi.org/10.30978/CEES-2025-2-48)
9. Cao S, Yin Y, Hu H, Hong S, He W, Lv W, et al. CircGLIS3 inhibits thyroid cancer invasion and metastasis through miR-146b-3p/AIF1L axis. *Cell Oncol (Dordr).* 2023;46(6):1777-89. doi: [10.1007/s13402-023-00845-2](https://doi.org/10.1007/s13402-023-00845-2)
10. Jia M, Shi Y, Li Z, Lu X, Wang J. MicroRNA-146b-5p as an oncomiR promotes papillary thyroid carcinoma development by targeting CCDC6. *Cancer Lett.* 2019;443:145-56. doi: [10.1016/j.canlet.2018.11.026](https://doi.org/10.1016/j.canlet.2018.11.026)
11. Silaghi CA, Lozovanu V, Silaghi H, Georgescu RD, Pop C, Dobrea A, et al. The Prognostic Value of MicroRNAs in Thyroid Cancers-A Systematic Review and Meta-Analysis. *Cancers (Basel).* 2020;12(9):2608. doi: [10.3390/cancers12092608](https://doi.org/10.3390/cancers12092608)
12. Papaioannou M, Chorti AG, Chatzikiyriakidou A, Giannoulis K, Bakkar S, Papavramidis TS. MicroRNAs in Papillary Thyroid Cancer: What Is New in Diagnosis and Treatment. *Front Oncol.* 2022;11:755097. doi: [10.3389/fonc.2021.755097](https://doi.org/10.3389/fonc.2021.755097)