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ORIGINAL ARTICLE

Bioinformatics-Based Analysis of Molecular Markers in Thyroid Cancer

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ABSTRACT

Background: **Thyroid cancer (TC) is a common endocrine malignancy for which additional molecular markers may improve biological understanding.**
Methods: **miRNA expression profiles from the GEO dataset GSE191117 were analyzed to identify differentially expressed miRNAs in thyroid carcinoma and normal thyroid tissues. Predicted targets of selected miRNAs were compared across miRWalk, TargetScan, and miRDB; Gene Ontology (GO) and KEGG enrichment analyses were performed.**
Results: **A total of 828 differentially expressed miRNAs were reported, and seven upregulated and five downregulated miRNAs were selected for target prediction. Enrichment analysis of predicted target genes implicated biological processes involving cell signaling, gene expression regulation, and development, including MAPK, PI3K-Akt, and ErbB signaling pathways.**
Conclusions: **Differentially expressed miRNAs and their predicted targets provide candidates for further study in thyroid cancer. These computational findings require independent experimental and clinical validation.**
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KEYWORDS

miRNA, thyroid cancer, differentially expressed miRNAs, predicted target genes

LIST OF ABBREVIATIONS

FNAB - Fine needle aspiration biopsy
GEO - Gene Expression Omnibus
GO - Gene Ontology
TC - Thyroid cancer
PTC - Papillary thyroid carcinoma
ATC - Anaplastic thyroid carcinoma
FTC - Follicular thyroid carcinoma
MTC - Medullary thyroid carcinoma
MAPK - Mitogen-activated protein kinase
PI3K/AKT - Phosphatidylinositol 3-kinase
FNAC - Fine-needle aspiration cytology
DEG - Differentially-expressed genes

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BP - Biological process
MF - Molecular function

INTRODUCTION

Thyroid cancer (TC) is a malignancy arising from the follicular or parafollicular cells within the thyroid gland. As a predominant malignancy within the endocrine system, TC constitutes approximately 1% of all diagnosed cancers globally. Despite its relatively low prevalence, epidemiological studies have demonstrated a significant upward trend in its incidence rates across diverse populations in recent decades [1]. TC is primarily categorized into four distinct histological subtypes: papillary, follicular, medullary, and anaplastic thyroid carcinomas [2]. Among the histological subtypes of thyroid malignancies, PTC demonstrates the highest prevalence, representing approximately four-fifths of all diagnosed cases. Following PTC in frequency, FTC constitutes a substantial proportion, ranging between 10 - 20% of total TC cases [3]. High-resolution thyroid ultrasonography, in conjunction with ultrasound-guided fineneedle aspiration cytology (FNAC), represents the cornerstone of thyroid nodule evaluation. Nevertheless, contemporary clinical studies reveal that approximately 25% of FNAC specimens fall into the category of cytological indeterminacy, presenting diagnostic dilemmas for clinicians [4]. Similar to other cancers, molecular markers in TC have gained prominence as valuable tools for advancing precision medicine strategies [5]. The past decade has witnessed significant advancements in deciphering the intricate molecular pathways underlying TC, particularly in the areas of genomic alterations, epigenetic modifications, and tumor microenvironment interactions. The malignant transformation of thyroid cells is primarily driven by dysregulation of two critical molecular pathways: the mitogen-activated protein kinase (MAPK) cascade and the PI3K-AKT-mTOR signaling axis [6]. Genomic analyses reveal that somatic BRAF mutations, particularly the V600E variant, are detectable in nearly half (45%) of thyroid carcinoma cases, representing one of the most prevalent oncogenic drivers in thyroid tumorigenesis [7,8]. Extensive molecular profiling has identified a unique miRNA expression pattern in PTC, marked by the pronounced overexpression of oncogenic miRNAs, such as the miR-221, miR-222, and miR-146b clusters, relative to surrounding normal thyroid tissue [9]. The aberrant expression patterns of these tumor-promoting miRNAs show a strong association with key clinicopathological indicators of disease advancement, including extracapsular invasion, metastatic dissemination to regional lymph nodes and distant organs, tumor recurrence patterns, and the presence of BRAFV600E oncogenic mutation [10,11]. Despite some progress in the study of the molecular mechanisms of TC, the molecular mechanisms of TC are still complex, and further exploratory studies of genomic, miRNA, and protein biomarkers are still needed. MicroRNAs (miRNAs) constitute a fundamental category of evolutionarily-conserved small regulatory molecules, typically spanning 19 - 25 nucleotides in length, which orchestrate post-transcriptional gene silencing through sequence-specific interactions with the matching sequences located in the 3′ untranslated segments of target messenger RNAs [12,13]. Emerging evidence positions miRNA expression profiling as a transformative diagnostic and prognostic tool in thyroid oncology, offering unprecedented potential for molecular stratification and personalized therapeutic strategies [14]. Five miRNAs, including miR-146, miR-221, miR-222, miR-21, and miR-181, were significantly upregulated in PTC compared to normal thyroid tissues. Among these, miR-146, miR-221, and miR-222 exhibited a remarkable 11- to 19-fold increase in expression levels in PTC relative to normal tissues [15]. Previous studies have revealed a potential link between elevated miR-146b levels and both extrathyroidal invasion and disease progression in papillary thyroid carcinoma [11]. Recent investigations have identified elevated expression levels of miR-34b, miR-130b, miR-146b, and miR-222, and in papillary thyroid carcinoma cases, exhibiting aggressive clinical behavior [16]. miRNAs are increasingly becoming notable non-coding RNAs in numerous thyroid cancer subtypes.

Microarray technology enables comprehensive analysis of miRNA expression profiles [17]. In this study, miRNA profiles from the GEO dataset GSE191117 were analyzed to identify differentially expressed miRNAs in thyroid tumor and normal thyroid tissues. Predicted target genes of selected miRNAs were compared across miRWalk, TargetScan, and miRDB, followed by Gene Ontology (GO) and KEGG pathway enrichment analysis to explore potential biological functions.

MATERIALS AND METHODS

Public data collection

The miRNA microarray data was retrieved from the Gene Expression Omnibus (GEO) database using the accession number GSE191117. The GSE191117 dataset comprises 111 formalin-fixed paraffin-embedded (FFPE) samples. Our analysis included miRNA profiles from 49 PTCs, 7 FTCs, 5 PTMCs, and 50 matched normal thyroid samples. These samples were dichotomized into a carcinoma group and a healthy control group for subsequent comparative analyses.

Differential expression analysis

Differential expression of miRNAs between normal thyroid tissues and TC tissues was performed using the GEO2R online analysis tool (https://www.ncbi.nlm.nih.gov/geo/geo2r/). The screening criteria were an adjusted p-value (adj.p) < 0.05 and an absolute log2 fold change (log2FC) ≥ 2. Based on the applied screening criteria, the miRNA exhibiting the greatest fold change was

Table 1. miRNAs with the highest multiplicity of differential expression in TC tissues and normal thyroid tissues.

Regulation	miRNA	Log2FC	adj. p-value	p-value
Upregulation	hsa-miR-146b-5p	2.403728	4.87E-06	1.18E-08
Upregulation	miR-15a-5p	0.482271	4.63E-03	2.79E-05
Upregulation	miR-221-3p	1.495657	2.31E-05	8.38E-08
Upregulation	miR-221-5p	1.12395	3.04E-04	1.47E-06
Upregulation	miR-222-3p	1.344468	2.33E-07	2.82E-10
Upregulation	miR-34a-5p	0.5679908	5.22E-03	3.78E-05
Upregulation	miR-551b-3p	1.063856	4.15E-02	6.02E-04
Downregulation	miR-100-5p	-0.4066661	2.06E-02	1.99E-04
Downregulation	miR-30a-3p	-0.4274567	2.44E-02	2.65E-04
Downregulation	miR-30a-5p	-0.4389135	4.15E-02	5.95E-04
Downregulation	miR-652-3p	-0.4478214	3.98E-02	4.80E-04
Downregulation	miR-7-5p	-1.468909	1.06E-02	8.94E-05

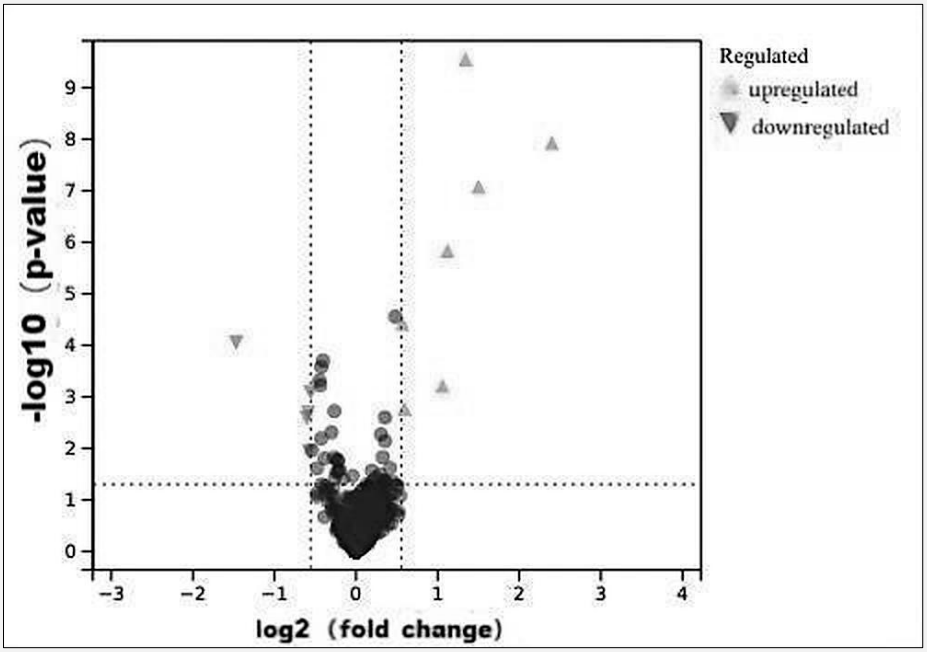


Figure 1. Volcano plot analysis of differentially expressed miRNAs in thyroid cancer and control samples.

identified.

Differential miRNA target gene prediction

In silico prediction of downstream targets was carried out for the seven upregulated and five downregulated miRNAs with the largest differential expression folds. To reduce the false-positive rate of software prediction results, it was formulated that at least one of the target genes in miRD-B (http://mirdb.org/), miRWalk (http://mirwalk.umm.uni-heidelberg.de/), and TargetScanHuman 7.2 (http: //www. targetscan.org/vert_72/), which are the trio of computational prediction tools, can be used only as target genes.

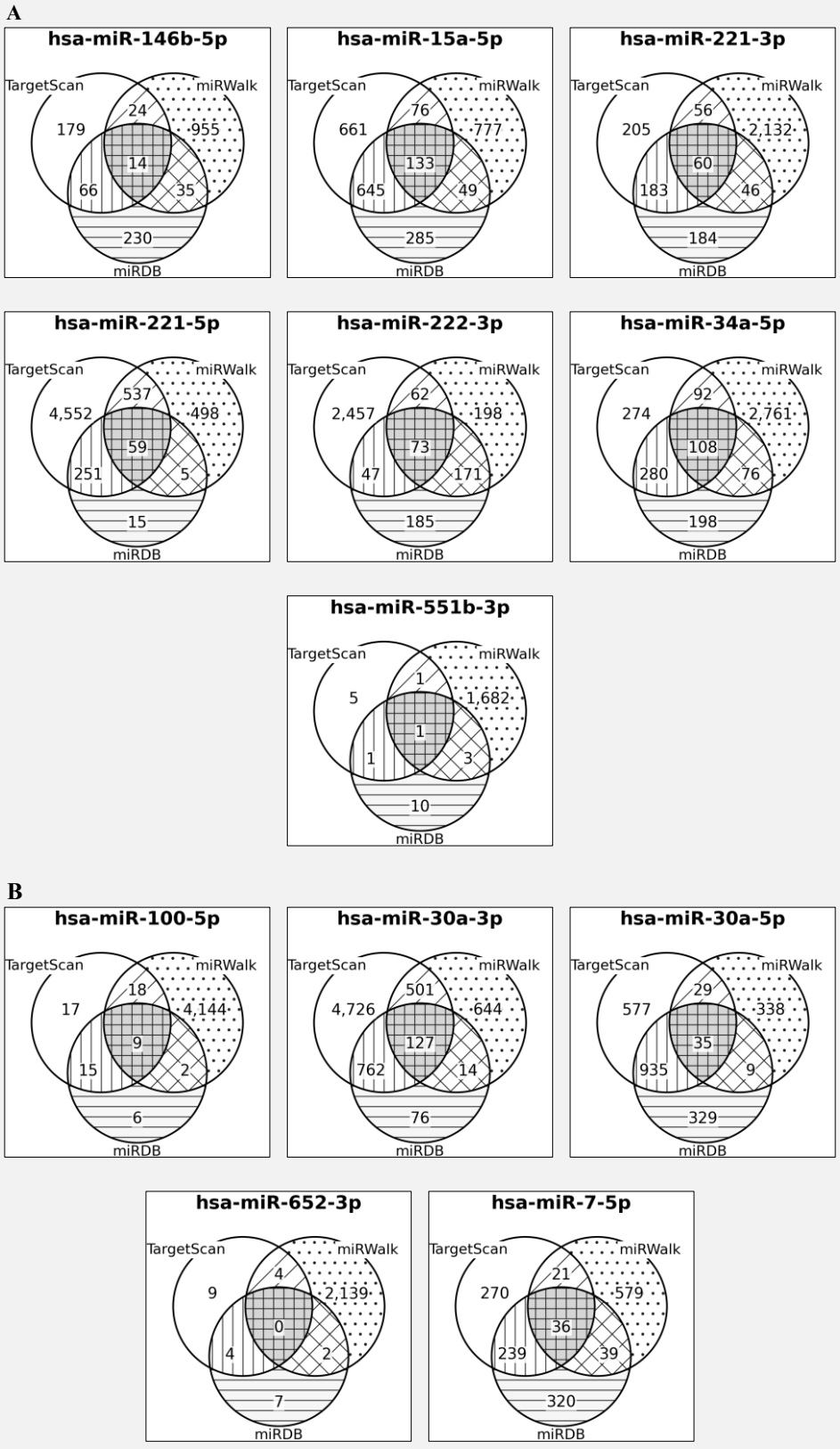
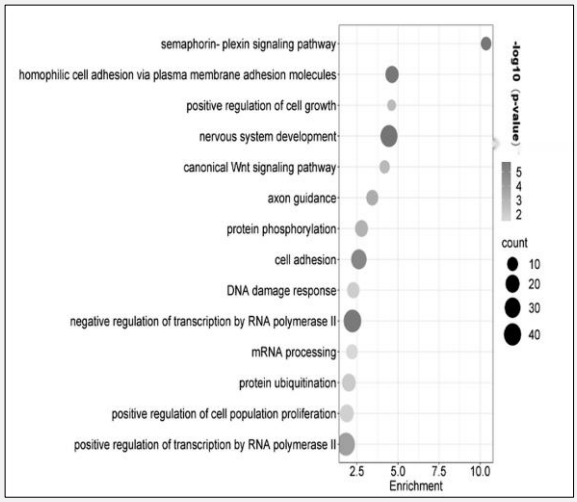
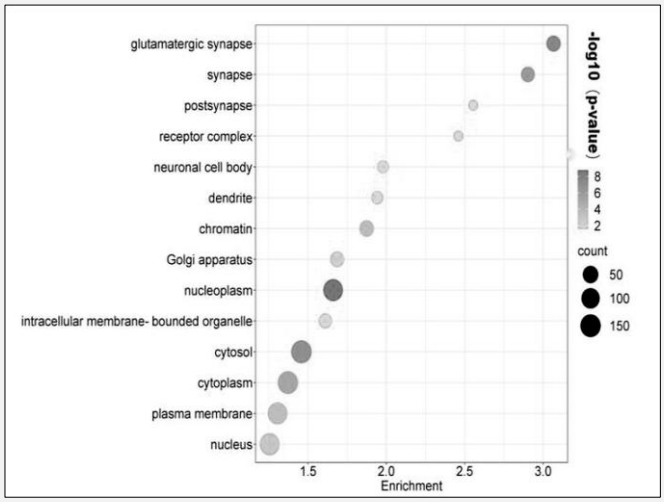
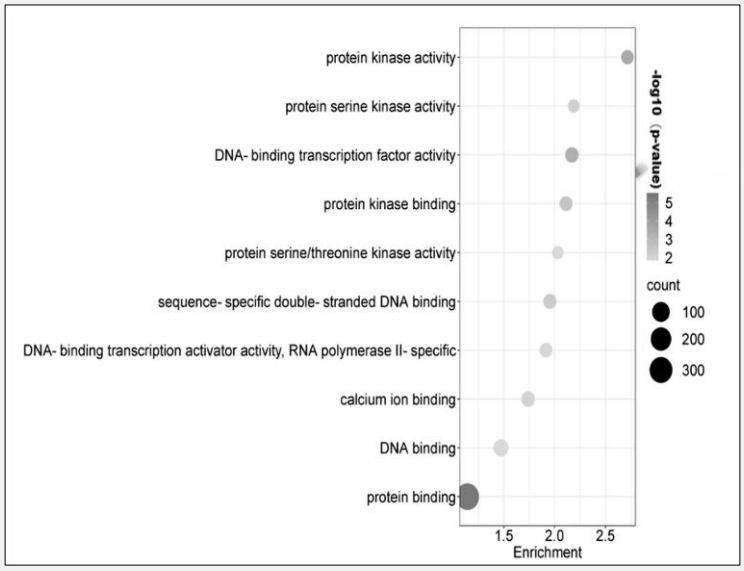


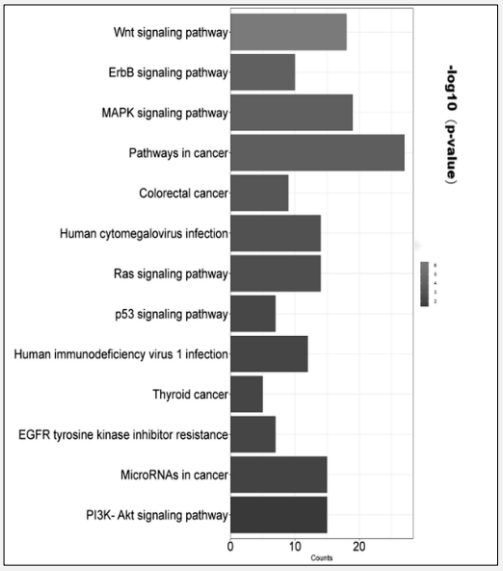
Figure 2. A: Indicate the number of target genes of miR-146b-5p, miR-15a-5p, miR-221-3p, miR-221-5p, miR-222-3p, miR-34a-5p, and miR-551b-3p in order. B: Indicate the number of target genes of miR-100-5p, miR-30a-3p, miR-30a-5p, miR-652-3p, and miR-7-5p in order.

A

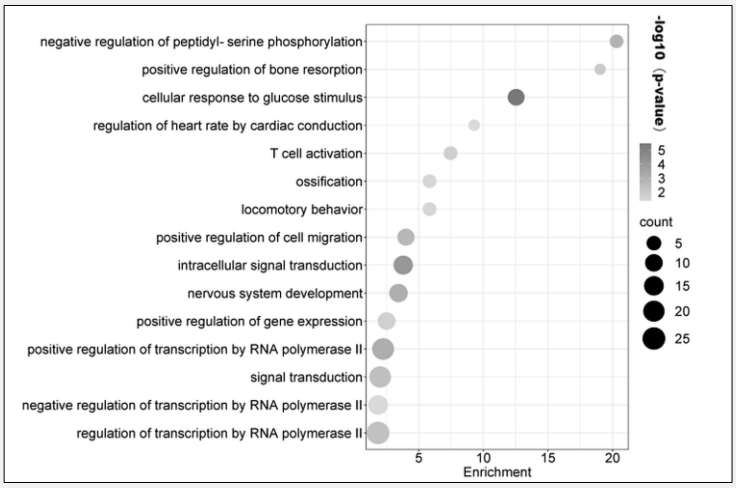

semaphorin- plexin signaling pathway
homophilic cell adhesion via plasma membrane adhesion molecules
positive regulation of cell growth
nervous system development
canonical Wnt signaling pathway
axon guidance
protein phosphorylation
cell adhesion
DNA damage response
negative regulation of transcription by RNA polymerase II
mRNA processing
protein ubiquitination
positive regulation of cell population proliferation
positive regulation of transcription by RNA polymerase II
2.5 5.0 7.5 10.0
Enrichment
-log10 (p-value)
5 4 3 2
count
10 20 30 40

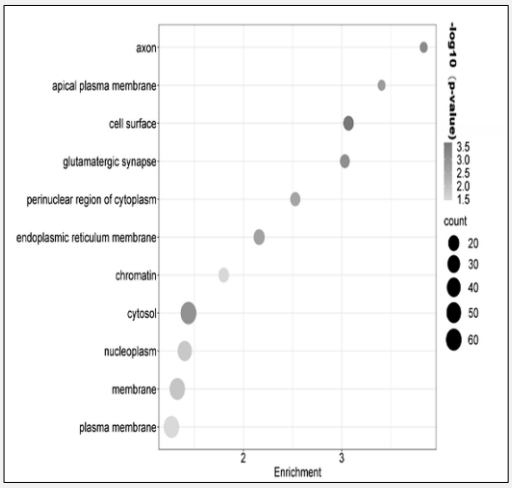

glutamatergic synapse
synapse
postsynapse
receptor complex
neuronal cell body
dendrite
chromatin
Golgi apparatus
nucleoplasm
intracellular membrane- bounded organelle
cytosol
cytoplasm
plasma membrane
nucleus
1.5 2.0 2.5 3.0
Enrichment
-log10 (p-value)
8 6 4 2
count
50 100 150


protein kinase activity
protein serine kinase activity
DNA- binding transcription factor activity
protein kinase binding
protein serine/threonine kinase activity
sequence- specific double- stranded DNA binding
DNA- binding transcription activator activity, RNA polymerase II- specific
calcium ion binding
DNA binding
protein binding
1.5 2.0 2.5
Enrichment
-log10 (p-value)
5 4 3 2
count
100 200 300


Wnt signaling pathway
ErbB signaling pathway
MAPK signaling pathway
Pathways in cancer
Colorectal cancer
Human cytomegalovirus infection
Ras signaling pathway
p53 signaling pathway
Human immunodeficiency virus 1 infection
Thyroid cancer
EGFR tyrosine kinase inhibitor resistance
MicroRNAs in cancer
PI3K- Akt signaling pathway
0 10 20
Counts
-log10 (p-value)

B


negative regulation of peptidyl- serine phosphorylation
positive regulation of bone resorption
cellular response to glucose stimulus
regulation of heart rate by cardiac conduction
T cell activation
ossification
locomotory behavior
positive regulation of cell migration
intracellular signal transduction
nervous system development
positive regulation of gene expression
positive regulation of transcription by RNA polymerase II
signal transduction
negative regulation of transcription by RNA polymerase II
regulation of transcription by RNA polymerase II
5 10 15 20
Enrichment
-log10 (p-value)
5 4 3 2
count
5 10 15 20 25


axon
apical plasma membrane
cell surface
glutamatergic synapse
perinuclear region of cytoplasm
endoplasmic reticulum membrane
chromatin
cytosol
nucleoplasm
membrane
plasma membrane
2 3
Enrichment
-log10 (p-value)
3.5 3.0 2.5 2.0 1.5
count
20 30 40 50 60

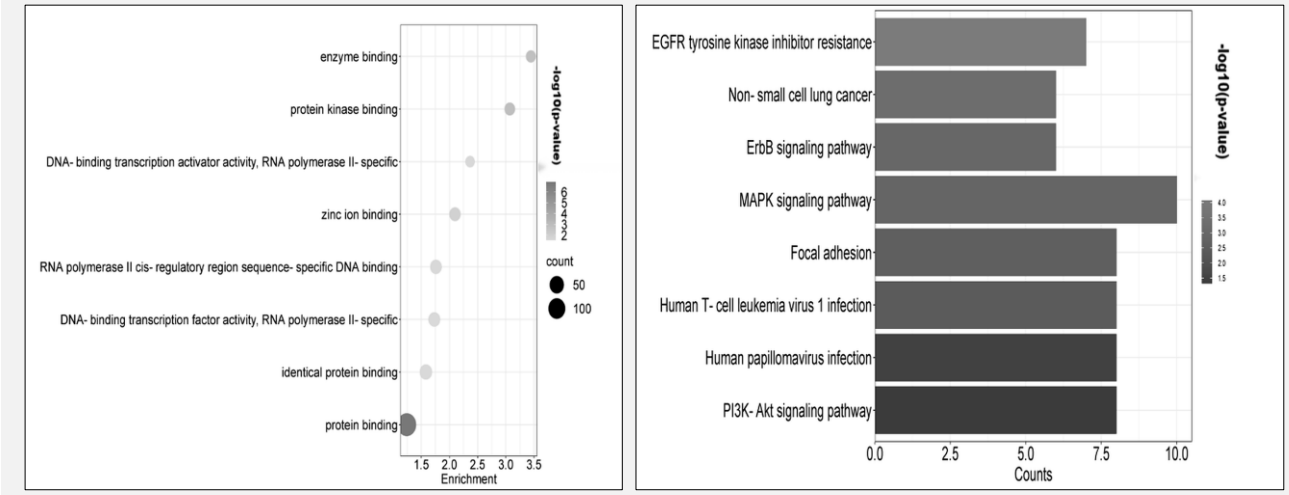


Figure 3. A: Upregulation of miRNA GO and KEGG analysis. B: Downregulation of miRNA GO and KEGG analysis p-values were less than 0.05.

Functional enrichment analysis of target genes
Functional characterization of predicted targets was conducted through Gene Ontology classification and KEGG pathway analysis utilizing the DAVID bioinformatics resource (https://david.ncifcrf.gov/). A significance threshold of $p < 0.05$ was applied, with multiple comparison adjustments performed using the Benjamini-Hochberg method.

RESULTS

Screening of differential miRNAs in TC
The GSE191117 dataset was analyzed using GEO2R, and a volcano plot of differentially expressed miRNAs was generated (Figure 1). A total of 828 differentially expressed miRNAs were identified. Seven upregulated and five downregulated miRNAs were selected for subsequent analysis. The seven upregulated miRNAs were miR-15a-5p, miR-146b-5p, miR-221-5p, miR-551b-3p, miR-221-3p, miR-34a-5p, and miR-222-3p. The five downregulated miRNAs were miR-30a-5p, miR-7-5p, miR-100-5p, miR-652-3p, and miR-30a-3p (Table 1).

Prediction of differential miRNA target genes
Candidate genes potentially regulated by differentially expressed miRNAs were identified through intersection analysis using three prediction algorithms: miRWalk, TargetScanHuman7.2, and miRDB. The analysis revealed distinct target gene profiles. Upregulated miRNAs, namely miR-146b-5p, miR-15a-5p, miR-221-3p, miR-221-5p, miR-222-3p, miR-34a-5p, and miR-551b-3p, targeted 14, 133, 60, 59, 73, 108 genes, and 1 gene, respectively (Figure 2A). Downregulated miRNAs, encompassing miR-100-5p, miR-30a-3p, miR-30a-5p, miR-652-3p, and miR-7-5p, targeted 9, 127, 35, 0, and 36 genes, respectively (Figure 2B).

Functional enrichment analysis
To reveal the functional implications of differentially expressed genes (DEGs), GO enrichment analysis was conducted utilizing the DAVID bioinformatics platform. Upregulated miRNAs were mostly implicated in biological processes (BP) including nervous system development and function, cell signaling, gene expression regulation, cell proliferation and migration, cell adhesion and development, protein modification and metabolism, etc. Cellular components (CC) were mainly focused on nucleus, cytoplasm, and cytosol, etc. Analysis of molecular functions (MF) revealed significant enrichment in transcriptional regulation processes, kinase-mediated phosphorylation, DNA-binding transcription factor operations, and various protein interaction activities, etc. KEGG pathway analysis revealed that target genes of upregulated miRNAs were primarily implicated in cancer-related pathways and Wnt signaling pathway, MAPK signaling pathway, PI3K-Akt signaling pathway, ErbB signaling pathway, etc. (Figure 3A). Downregulated miRNAs were primarily associated with cell signaling and metabolism, gene expression regulation, nervous system development and function, cell proliferation and migration, tissue and organ development, immunity and behavior, metabolism and physiology, protein modification and metabolism, cardiovascular func-

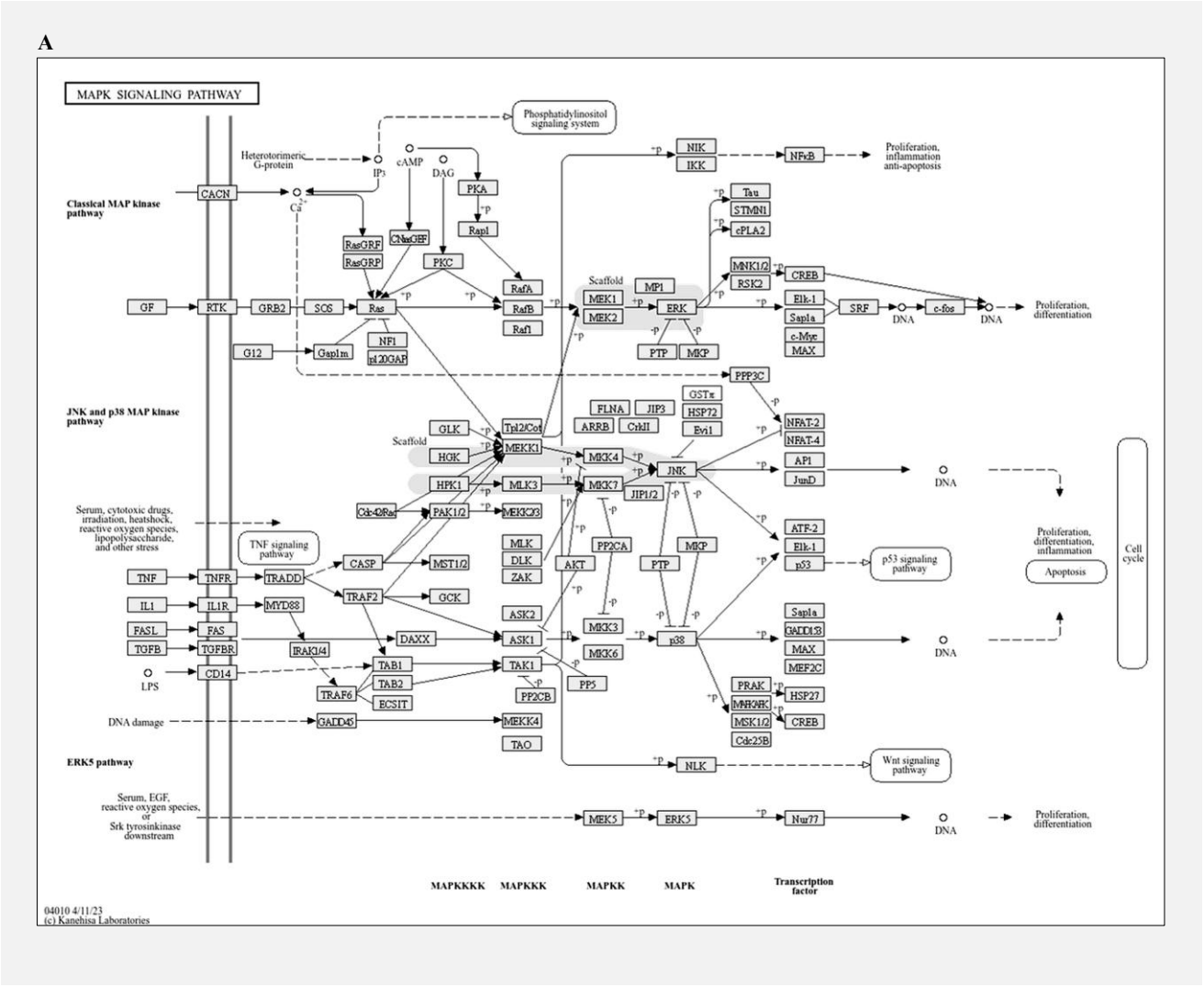


Figure 4A.

tion, and metabolism, etc. CC was mainly localized in axon, apical plasma membrane, cell surface, cytoplasm and nucleus, etc. Molecular function (MF) analysis mainly showed enrichment in enzyme binding, protein kinase binding, protein binding, etc. Downregulated miRNAs mainly targeted genes that were significantly enriched in the MAPK signaling pathway, human papillomavirus infection, and the PI3K-Akt signaling pathway (Figure 3B). The most important pathways, MAPK signaling pathway (Figure 4A) and PI3K-Akt signaling pathway (Figure 4B), are shown.

DISCUSSION

Analysis of the GSE191117 miRNA expression dataset identified 828 differentially expressed miRNAs between thyroid cancer and control samples. Of these, seven upregulated and five downregulated miRNAs were selected for subsequent target prediction. The observed expression differences suggest that miRNA dysregulation is associated with thyroid cancer, although expression profiling alone does not establish causality or clinical diagnostic performance.

Predicted targets were compared using miRWalk, TargetScan, and miRDB, and the resulting gene sets were analyzed by GO and KEGG enrichment. The functional annotations implicated processes involving gene expression regulation, cell signaling, development, and protein-related functions. Enriched pathways included MAPK, PI3K-Akt, ErbB, and other cancer-associated signaling pathways (Figures 3 and 4). These findings should be interpreted as associations derived from computational prediction and enrichment, rather than direct evidence that these pathways are activated or inhibited in the analyzed tissues.

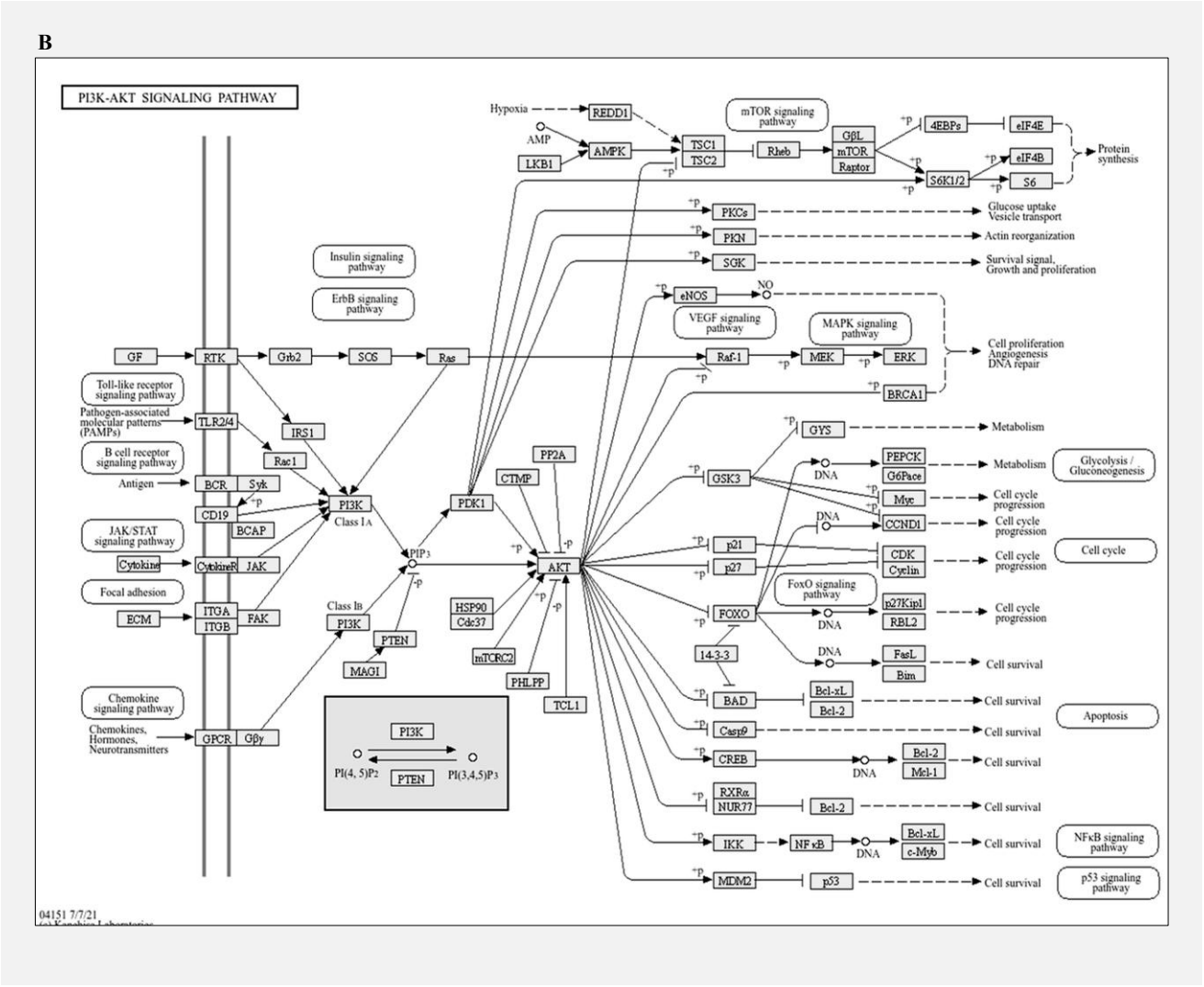


Figure 4. Pathway enrichment analysis of universal DEG.

A: Insights into TC-related pathways (HSA04010: MAPK signaling pathway). B: Insights into TC-related pathways (HSA04151: PI3K-Akt signaling pathway).

The differential expression of miRNAs such as miR-146b, miR-221, and miR-222 is consistent with previous investigations of thyroid tumors [11,16,18-21]. Published work has also examined differences in miRNA expression among thyroid cancer subtypes and the potential utility of miR-7 and miR-221/222 as candidate biomarkers [22-24]. In the present study, however, no experimental validation of individual miRNA-target gene interactions or diagnostic accuracy was performed. Therefore, the candidate miRNAs and their predicted targets require independent validation before they can be considered clinically useful biomarkers or therapeutic targets.

The present analysis has several limitations. The selection of target genes was based on computational prediction, which can yield false-positive relationships, and functional enrichment does not demonstrate a direct mechanistic effect. In addition, the composition and size of the available cohort may limit generalizability. Future studies should validate miRNA expression and predicted targets in independent cohorts and investigate relevant biological pathways through experimental approaches.

CONCLUSION

In conclusion, analysis of the GSE191117 dataset identified differentially expressed miRNAs in thyroid cancer samples. Target prediction using three databases and functional enrichment analysis suggested that the predicted targets may participate in cancer-associated bio-

logical processes and signaling pathways, including MAPK and PI3K-Akt signaling. These findings are hypothesis-generating and may guide further investigation of candidate miRNAs and their target genes; their biological functions and clinical relevance require independent validation.

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Availability of Data and Materials:
The data supporting the results of this study are available in the GEO database (https://www.ncbi.nlm.nih.gov/geo/).

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Declaration of Generative AI in Scientific Writing:
The authors declare that ChatGPT (OpenAI) was used in this revision solely for AI-assisted language editing, including grammar correction and language checking. ChatGPT was not used to generate scientific content, analyze data, interpret results, or draw conclusions. The authors reviewed and edited all AI-assisted text and take full responsibility for the content of the manuscript.

Declaration of Interest:
The authors state no conflict of interest. All authors agreed to the submission, and they confirm that the manuscript has not been published in whole or in parts nor has it been submitted anywhere else.

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