Cancer Screening and Prevention **2024** vol. 3(4) | 198–202
DOI: 10.14218/CSP.2024.00024

Original Article

Absence of Association Between the *miR-27a* rs895819 T>C Polymorphism and Susceptibility to Wilms Tumor

Shuang Wu, Changmi Deng, Yufeng Han, Wen Fu and Ruixi Hua*

Department of Pediatric Surgery, Guangzhou Institute of Pediatrics, Guangdong Provincial Key Laboratory of Research in Structural Birth Defect Disease, Guangzhou Women and Children's Medical Center, Guangzhou Medical University, Guangzhou, Guangdong, China

Received: October 21, 2024 | **Revised:** December 04, 2024 | **Accepted:** December 17, 2024 | **Published online:** December 30, 2024

Abstract

Background and objectives: Wilms tumor is the most common kidney tumor in children aged 0-14 years. MicroRNAs are small, noncoding RNAs linked to the development of malignant tumors. Several studies have shown the association between single nucleotide polymorphism in *miR-27a* and cancer risk. This study aimed to explore the potential impact of the *miR-27a* rs895819 T>C polymorphism on Wilms tumor susceptibility.

Methods: The rs895819 T>C polymorphism was genotyped using the TaqMan method in 145 patients with Wilms tumors and 531 controls. Logistic regression models were used to assess the association between this polymorphism and Wilms tumor risk. A stratified analysis was also performed based on age, sex, and clinical stage.

Results: The rs895819 T>C polymorphism showed genotypic distribution consistent with Hardy-Weinberg equilibrium (P = 0.749). The differences were not statistically significant. The *miR-27a* rs895819 T>C polymorphism was not significantly associated with Wilms tumor susceptibility, and the stratified analysis did not yield any significant differences.

Conclusions: Our study provides evidence of a lack of association between the *miR-27a* rs895819 T>C polymorphism and Wilms tumor susceptibility. Further validation through larger sample sizes and additional genetic polymorphisms is warranted.

Introduction

Wilms tumor is a cancerous kidney tumor that originates from embryonic cells. It is the most prevalent kidney tumor among children aged 0–14 years globally, representing 90% of kidney tumors, with an incidence rate of 3.9 per million.[1] Most Wilms tumor patients present with isolated nephroblastoma, while 5–7% exhibit bilateral renal involvement, and 10% have multiple lesions within a single kidney. Nephrogenic rests (NRs), which retain the potential to form embryonic tumors, are considered Wilms tumor precursors. These occur after 36 weeks of gestation and persist as embryonic cells or tissues after birth.[2] Notably, NRs play a crucial role in Wilms tumor diagnosis. Approximately 40% of unilateral Wilms tumors and over 90% of bilateral Wilms tumors are accompanied by NRs.[3]

The cumulative death rate from various causes, excluding recurrence, reaches 5.4% 30 years after Wilms tumor diagnosis, increasing significantly to 22.7% by the 50-year mark.[4] At the 50-year milestone, subsequent primary neoplasms and heart disease contribute to cumulative mortality rates of 8.2% and 6.3%, respectively.[4] The Renal Tumor Study Group of the International Society of Paediatric Oncology system guides treatment decisions for Wilms tumor. Stage I tumors remain confined to the renal capsule, while stage II tumors infiltrate the renal sinuses, perirenal fat, and adjacent organs without a discernible margin but with a well-defined excision margin. Prechemotherapy-induced tumor rupture is diagnosed through imaging, and chemotherapy-induced lymph node changes serve as stage III indicators. Stage IV tumors involve hematogenous metastases or lymph node metastases beyond the abdominal area.[5]

Genetic syndromes such as Denys-Drash syndrome and WAGR syndrome (Wilms tumor, absence of the iris, genitourinary abnormalities, and development retardation), are linked to a greater likelihood of developing Wilms tumor.[6] The Children's Oncology Group of America program incorporates molecular markers, including blastemal volume and 1q gain, into clinical decision-

Keywords: Wilms tumor; *miR-27a*; rs895819; Polymorphism; Susceptibility; Chinese children.

***Correspondence to:** Ruixi Hua, Department of Pediatric Surgery, Guangzhou Institute of Pediatrics, Guangdong Provincial Key Laboratory of Research in Structural Birth Defect Disease, Guangzhou Women and Children's Medical Center, Guangzhou Medical University, 9 Jinsui Road, Guangzhou, Guangdong 510623, China. ORCID: https://orcid.org/0000-0001-5319-656X. Tel: +86-020-38076560, E-mail: huaruix@mail.sysu.edu.cn

How to cite this article: Wu S, Deng C, Han Y, Fu W, Hua R. Absence of Association Between the *miR-27a* rs895819 T>C Polymorphism and Susceptibility to Wilms Tumor. *Cancer Screen Prev* 2024;3(4):198–202. doi: 10.14218/CSP.2024.00024.

Table 1. Genotype distributions of *miR-27a* rs895819 T>C polymorphism and Wilms tumor susceptibility

Genotype	Cases (n = 145)	Controls (n = 531)	*P*[a]	Crude OR (95% CI)	*P*	Adjusted OR (95% CI)[b]	*P*[b]
rs895819 (HWE = 0.749)							
TT	72 (49.66)	285 (53.67)		1.00		1.00	
TC	64 (44.14)	210 (39.55)		1.21 (0.82–1.77)	0.335	1.20 (0.82–1.75)	0.358
CC	9 (6.21)	36 (6.78)		0.99 (0.46–2.15)	0.979	0.96 (0.44–2.09)	0.923
Additive			0.608	1.09 (0.82–1.47)	0.552	1.08 (0.81–1.45)	0.604
Dominant	73 (50.34)	246 (46.33)	0.391	1.18 (0.81–1.70)	0.391	1.16 (0.80–1.68)	0.425
TT/TC	136 (93.79)	495 (93.22)		1.00		1.00	
CC	9 (6.21)	36 (6.78)	0.806	0.91 (0.43–1.94)	0.806	0.89 (0.42–1.89)	0.757
T	208 (71.72)	780 (73.45)		1.00		1.00	
C	82 (28.28)	282 (26.55)	0.558	1.09 (0.82–1.46)	0.558	1.08 (0.81–1.44)	0.610

[a]χ^2 test for genotype distributions between Wilms tumor patients and controls. [b]Adjusted for age and gender. CI, confidence interval; HWE, Hardy-Weinberg equilibrium; OR, odds ratio.

making.[7] Additionally, a study revealed that altered microRNA (miRNA) expression patterns correlate with a poor prognosis in the blast histology of pediatric Wilms tumor patients treated with chemotherapy.[8,9]

miRNAs, a class of small noncoding RNAs, play a critical role in cancer risk. Case-control studies have shown that polymorphisms in miRNA-encoding genes are associated with malignant tumors.[10,11] Recent findings have suggested that *miR-27a* in serum/plasma or tumor tissue can serve as a diagnostic biomarker and predict the survival of cancer patients.[12] Northern blot analysis has confirmed *miR-27a* expression in mouse kidneys.[13] Additionally, a recent study showed that *miR-27a-3p* expression is associated with the grade of malignancy in Wilms tumor.[14] A single-nucleotide polymorphism (SNP) is a genetic variation that involves a change in a single nucleotide at a specific location within the genome. A case-control study highlighted the protective effects of the *miR-27a* rs895819 T>C polymorphism against cervical cancer.[15] Conversely, a meta-analysis linked the *miR-27a* rs895819 T>C polymorphism to an increased risk of colorectal cancer in the Chinese population.[16] However, no reports have explored the relationship between the *miR-27a* rs895819 T>C polymorphism and Wilms tumor susceptibility. Therefore, this case-control study aimed to elucidate this association.

Materials and methods

Study population

We included 145 patients with Wilms tumors as the case group and 531 cancer-free patients as the control group (Table S1).[17–20] Both the case group and the control group were recruited from Guangzhou Women and Children's Medical Center. The patients in the case group were diagnosed with Wilms tumor at the hospital and did not have any history of other malignant tumors. These two groups were compared based on age and sex, while the case group was categorized according to clinical stage. This study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). This research was approved by the institutional review committee of Guangzhou Women and Children's Medical Center (Approval No. 202016601). All participants or their legal guardians provided informed written consent.

SNP selection and genotyping

Total genomic DNA was extracted from paraffin-embedded tissues and peripheral blood using the QIAamp DNA FFPE Tissue Kit (Qiagen, Valencia, CA) and the TIANGEN Blood DNA Extraction Kit (TianGen Biotech, Beijing), respectively. The *miR-27a* rs895819 T>C polymorphism was genotyped using the TaqMan method and the 7900 sequencing detection system (Applied Biosystems, Foster City, CA, USA). Further methodological details are provided in the cited studies.[21–24] To ensure genotyping accuracy, 10% of the samples were retested, and the results were consistent with previous results.

Statistical analysis

Various statistical methods were employed based on variable types. The χ^2 test was utilized to evaluate potential disparities between patients and controls. Additionally, the χ^2 goodness-of-fit test was utilized to evaluate whether the selected SNP deviated from Hardy-Weinberg equilibrium in the control group. Demographic variables and gene frequencies across subjects were analyzed using two-sided χ^2 tests. Multivariate logistic regression analysis was performed to calculate adjusted odds ratios (ORs) and 95% confidence intervals (CIs) to examine the relationship between the rs895819 T>C polymorphism and susceptibility to Wilms tumor. All analyses were performed using the SAS statistical package (version 9.4; SAS Institute, USA), with statistical significance set at $P < 0.05$.

Results

Association of the miR-27a polymorphism with Wilms tumor susceptibility

The *miR-27a* rs895819 T>C polymorphism was successfully detected in 145 patients and 531 controls. As presented in Table 1, the genotype distribution of the *miR-27a* rs895819 T>C polymorphism in both the case and control groups adhered to Hardy-Weinberg equilibrium ($P = 0.749$). No statistically significant differences were observed between the case and control groups (TC vs. TT: adjusted OR = 1.20, 95% CI = 0.82–1.75, $P = 0.358$; CC vs. TT: adjusted OR = 0.96, 95% CI = 0.44–2.09, $P = 0.923$; TT vs. TT/TC: adjusted OR = 1.16, 95% CI = 0.80–1.68, $P = 0.425$; CC vs. TT/TC: adjusted OR = 0.89, 95% CI = 0.42–1.89, $P = 0.757$;

Table 2. Stratification analysis for the association of *miR-27a* rs895819 T>C polymorphism with Wilms tumor risk

Variables	TT[a] (Cases/Controls)	TC/CC[a] (Cases/Controls)	Crude OR[a] (95% CI)	*P*	Adjusted OR[aa] (95% CI)	*P*[a]
Age, month						
≤18	32/120	34/113	1.13 (0.65–1.95)	0.665	1.11 (0.64–1.93)	0.703
>18	40/165	39/133	1.21 (0.74–1.99)	0.453	1.21 (0.74–2.00)	0.444
Gender						
Females	34/129	30/104	1.09 (0.63–1.91)	0.750	1.09 (0.63–1.91)	0.751
Males	38/156	43/142	1.24 (0.76–2.03)	0.386	1.21 (0.74–1.98)	0.456
Clinical stage						
I+II	25/285	28/246	1.30 (0.74–2.28)	0.367	1.29 (0.73–2.29)	0.383
III+IV	41/285	42/246	1.19 (0.75–1.89)	0.468	1.19 (0.75–1.89)	0.468

[a]Adjusted for age and gender. CI, confidence interval; OR, odds ratio.

C vs. T: adjusted OR = 1.08, 95% CI = 0.81–1.44, *P* = 0.610). We failed to establish a significant association between the *miR-27a* rs895819 T>C polymorphism and Wilms tumor susceptibility.

Stratification analysis

We conducted a stratification analysis to investigate the association between the rs895819 T>C polymorphism and the risk of Wilms tumor while, taking into account age, sex, and clinical stage (Table 2). The results showed that age (≤18, adjusted OR = 1.11, 95% CI = 0.64–1.93, *P* = 0.703; >18, adjusted OR = 1.21, 95% CI = 0.74–2.00, *P* = 0.444), sex (female, adjusted OR = 1.09, 95% CI = 0.63–1.91, *P* = 0.751; male, adjusted OR = 1.21, 95% CI = 0.74–1.98, *P* = 0.456), and clinical stage (I+II, adjusted OR = 1.29, 95% CI = 0.73–2.29, *P* = 0.383; III+IV, adjusted OR = 1.19, 95% CI = 0.75–1.89, *P* = 0.468) were not significantly associated with the miR-27a rs895819 T>C polymorphism in relation to Wilms tumor susceptibility, even after stratification.

Discussion

In this study, we investigated the association between the *miR-27a* rs895819 T>C polymorphism and Wilms tumor risk. We found no significant difference in the association between *miR-27a* rs895819 T>C and Wilms tumor risk.

As a class of noncoding RNA molecules, miRNAs play a pivotal role in negatively regulating the expression of multiple protein-coding genes. They also modulate various cancer-related genes at the posttranscriptional level.[25] Recently, miRNA SNPs have garnered attention for their potential association with tumor risk. These SNPs have emerged as candidate biomarkers for breast cancer susceptibility.[26,27] According to comprehensive Genome-Wide Association Studies, 26 miRNA SNPs were significantly linked to susceptibility to colorectal cancer, other cancers, obesity, and celiac disease.[28] These findings suggest that miRNA SNPs could be targeted in Genome-Wide Association Studies to identify potential susceptibility loci.

Multiple studies have provided evidence for the close association between *miR-27a*, a small RNA molecule, and cancer susceptibility, including but not limited to renal cell carcinoma, gastric cancer, and colorectal cancer.[29,30] Notably, *miR-27a* expression is downregulated in high-risk Wilms tumors.[31] Furthermore, research has shown that *miR-27a-5p* significantly inhibits Wilms tumor development by suppressing the expression of *PBOV1*.[32] Conversely, in renal cancer cells, *miR-27a* suppresses growth by targeting *ERFR*.[33] Interestingly, *miR-27a* upregulation in *B4GALT3* has been associated with cervical cancer progression and may serve as a potential biomarker for this disease.[34] Additionally, proteomic analysis has linked elevated levels of *miR-27a* to distant metastasis and poor prognosis in colorectal cancer patients.[35] These diverse effects of *miR-27a* across different malignant tumors likely result from a combination of factors.

The *miR-27a* rs895819 T>C SNP is located on chromosome 19p13.12 and plays a crucial role in regulating gene polymorphisms, tumorigenesis, proliferation, and apoptosis. While some studies have suggested that the *miR-27a* rs895819 polymorphism increases the risk of gastric cancer, others have found no association with gastric epithelial dysplasia.[36] In a case-control study, the *miR-27a* rs895819 polymorphism was found to be predictive of renal cell carcinoma risk in the Chinese population.[37] We did not find any significant association between the *miR-27a* rs895819 T>C polymorphism and Wilms tumor risk in our study. However, we observed that the risk of disease tends to decrease linearly with the addition of recessive mutated genes, even if no significant difference was detected. The negative results in this study could be attributed to various factors, including limitations in sample size, diversity among subject groups, geographic variations, genetic background, or potential errors in analysis.

This study faces several challenges that must be addressed for more accurate results. First, Wilms tumor is a complex disease involving multiple genes and intricate interactions. Relying solely on gene polymorphism analysis may lead to inaccurate negative results. Second, increasing the sample size in the case group is essential for establishing a robust correlation between variables. Finally, potential biases during data acquisition and analysis must be carefully considered to ensure the accuracy and reliability of our findings.

Conclusions

Our study provides evidence that there is no association between the *miR-27a* rs895819 T>C polymorphism and Wilms tumor susceptibility. To validate these findings, further investigations should focus on expanding the sample size and exploring additional genetic polymorphisms.

Acknowledgments

None.

Funding

This work was supported by grants from the National Natural Science Foundation of China (No. 82003523), Guangzhou Science and Technology Project (No. SL2024A04J02146, SL2024A03J01616).

Conflict of interest

The authors have no conflicts of interest related to this publication.

Author contributions

Investigation, manuscript writing (SW), investigation, sample and clinical information collection (CD, YH, WF), conception, data collection, and data analysis (RH). All authors have significantly contributed to the study and have given their approval to the final draft of the manuscript.

Ethical statement

This study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). This research was approved by the institutional review committee of Guangzhou Women and Children's Medical Center (Approval No. 202016601). All participants or their legal guardians provided informed written consent.

Data sharing statement

The requested data can be readily accessed by reaching out to the corresponding author.

References

[1] Nakata K, Colombet M, Stiller CA, Pritchard-Jones K, Steliarova-Foucher E, IICC-3 Contributors. Incidence of childhood renal tumours: An international population-based study. Int J Cancer 2020;147(12):3313–3327. doi:10.1002/ijc.33147, PMID:32902866.

[2] Beckwith JB, Kiviat NB, Bonadio JF. Nephrogenic rests, nephroblastomatosis, and the pathogenesis of Wilms' tumor. Pediatr Pathol 1990;10(1-2):1–36. doi:10.3109/15513819009067094, PMID:2156243.

[3] Vujanić GM, Sandstedt B. The pathology of Wilms' tumour (nephroblastoma): the International Society of Paediatric Oncology approach. J Clin Pathol 2010;63(2):102–109. doi:10.1136/jcp.2009.064600, PMID:19687012.

[4] Wong KF, Reulen RC, Winter DL, Guha J, Fidler MM, Kelly J, *et al*. Risk of Adverse Health and Social Outcomes Up to 50 Years After Wilms Tumor: The British Childhood Cancer Survivor Study. J Clin Oncol 2016;34(15):1772–1779. doi:10.1200/JCO.2015.64.4344, PMID:27022116.

[5] Vujanić GM, Gessler M, Ooms AHAG, Collini P, Coulomb-l'Hermine A, D'Hooghe E, *et al*. The UMBRELLA SIOP-RTSG 2016 Wilms tumour pathology and molecular biology protocol. Nat Rev Urol 2018;15(11):693–701. doi:10.1038/s41585-018-0100-3, PMID:30310143.

[6] Fischbach BV, Trout KL, Lewis J, Luis CA, Sika M. WAGR syndrome: a clinical review of 54 cases. Pediatrics 2005;116(4):984–988. doi:10.1542/peds.2004-0467, PMID:16199712.

[7] Huang X, Zhao J, Zhu J, Chen S, Fu W, Tian X, *et al*. MYCN gene polymorphisms and Wilms tumor susceptibility in Chinese children. J Clin Lab Anal 2019;33(9):e22988. doi:10.1002/jcla.22988, PMID:31343784.

[8] Wegert J, Ishaque N, Vardapour R, Geörg C, Gu Z, Bieg M, *et al*. Mutations in the SIX1/2 pathway and the DROSHA/DGCR8 miRNA microprocessor complex underlie high-risk blastemal type Wilms tumors. Cancer Cell 2015;27(2):298–311. doi:10.1016/j.ccell.2015.01.002, PMID:25670083.

[9] de Sá Pereira BM, Montalvão de Azevedo R, da Silva Guerra JV, Faria PA, Soares-Lima SC, De Camargo B, *et al*. Non-coding RNAs in Wilms' tumor: biological function, mechanism, and clinical implications. J Mol Med (Berl) 2021;99(8):1043–1055. doi:10.1007/s00109-021-02075-1, PMID:33950291.

[10] Dai J, Chen Y, Gong Y, Gu D, Chen J. Association of microRNA-27a rs895819 polymorphism with the risk of cancer: An updated meta-analysis. Gene 2020;728:144185. doi:10.1016/j.gene.2019.144185, PMID:31838252.

[11] Li X, Xu M, Ding L, Tang J. MiR-27a: A Novel Biomarker and Potential Therapeutic Target in Tumors. J Cancer 2019;10(12):2836–2848. doi:10.7150/jca.31361, PMID:31258791.

[12] Bi W, Li J, Xiong M, Pan B, Zhang Z, Nasifu L, *et al*. The diagnostic and prognostic role of miR-27a in cancer. Pathol Res Pract 2023;247:154544. doi:10.1016/j.prp.2023.154544, PMID:37235911.

[13] Lagos-Quintana M, Rauhut R, Lendeckel W, Tuschl T. Identification of novel genes coding for small expressed RNAs. Science 2001;294(5543):853–858. doi:10.1126/science.1064921, PMID:11679670.

[14] Wang X, Li X, Tan L, Zhang F, Zhang J, Zhao X, *et al*. Identification and Validation of Lipid Metabolism Gene FASN-Associated miRNA in Wilms Tumor. Biochem Genet 2024. doi:10.1007/s10528-024-10703-x, PMID:38416272.

[15] Chen G, Zhang M, Zhu J, Chen F, Yu D, Zhang A, *et al*. Common genetic variants in pre-microRNAs are associated with cervical cancer susceptibility in southern Chinese women. J Cancer 2020;11(8):2133–2138. doi:10.7150/jca.39636, PMID:32127940.

[16] Yuan L, Zhang TT, Ren Y. miR-27a rs895819 polymorphism and risk of cancer in Chinese population: a meta-analysis. J Evid Based Med 2015;8(2):75–83. doi:10.1111/jebm.12152, PMID:25976406.

[17] Fu W, Zhu J, Xiong SW, Jia W, Zhao Z, Zhu SB, *et al*. BARD1 Gene Polymorphisms Confer Nephroblastoma Susceptibility. EBioMedicine 2017;16:101–105. doi:10.1016/j.ebiom.2017.01.038, PMID:28161399.

[18] Fu W, Li L, Xiong SW, Zhang T, Jia W, Zhu J, *et al*. miR-423 rs6505162 C>A polymorphism contributes to decreased Wilms tumor risk. J Cancer 2018;9(14):2460–2465. doi:10.7150/jca.24916, PMID:30026843.

[19] Zhu J, Fu W, Jia W, Xia H, Liu GC, He J. Association between NER Pathway Gene Polymorphisms and Wilms Tumor Risk. Mol Ther Nucleic Acids 2018;12:854–860. doi:10.1016/j.omtn.2018.08.002, PMID:30161024.

[20] Zhu J, Jia W, Wu C, Fu W, Xia H, Liu G, *et al*. Base Excision Repair Gene Polymorphisms and Wilms Tumor Susceptibility. EBioMedicine 2018;33:88–93. doi:10.1016/j.ebiom.2018.06.018, PMID:29937070.

[21] Wang WJ, Zhou CL, Zhang XX, Zhao YM, Deng CM, Wu HY, *et al*. Association Between miR-492 rs2289030 G>C and Susceptibility to Neuroblastoma in Chinese Children from Jiangsu Province. Cancer Screen Prev 2023;2(4):199–203. doi:10.14218/CSP.2023.00025S.

[22] He J, Zou Y, Liu X, Zhu J, Zhang J, Zhang R, *et al*. Association of Common Genetic Variants in Pre-microRNAs and Neuroblastoma Susceptibility: A Two-Center Study in Chinese Children. Mol Ther Nucleic Acids 2018;11:1–8. doi:10.1016/j.omtn.2018.01.003, PMID:29858046.

[23] Guan Q, Lin H, Hua W, Lin L, Liu J, Deng L, *et al*. Variant rs8400 enhances ALKBH5 expression through disrupting miR-186 binding and promotes neuroblastoma progression. Chin J Cancer Res 2023;35(2):140–162. doi:10.21147/j.issn.1000-9604.2023.02.05, PMID:37180836.

[24] Chen YP, Liao YX, Zhuo ZJ, Yuan L, Lin HR, Miao L, *et al*. Association between genetic polymorphisms of base excision repair pathway and glioma susceptibility in Chinese children. World J Pediatr 2022;18(9):632–635. doi:10.1007/s12519-022-00562-0, PMID:35543809.

[25] Latini A, Ciccacci C, Novelli G, Borgiani P. Polymorphisms in miRNA genes and their involvement in autoimmune diseases susceptibility. Immunol Res 2017;65(4):811–827. doi:10.1007/s12026-017-8937-8, PMID:28741258.

[26] Hua HB, Yan TT, Sun QM. miRNA polymorphisms and risk of gastric

cancer in Asian population. World J Gastroenterol 2014;20(19):5700–5707. doi:10.3748/wjg.v20.i19.5700, PMID:24914331.
[27] Jia Z, Huang Y, Liu J, Liu G, Li J, Xu H, *et al*. Single nucleotide polymorphisms associated with female breast cancer susceptibility in Chinese population. Gene 2023;884:147676. doi:10.1016/j.gene.2023.147676, PMID:37524136.
[28] Shi TY, Zhu ML, He J, Wang MY, Li QX, Zhou XY, *et al*. Polymorphisms of the Interleukin 6 gene contribute to cervical cancer susceptibility in Eastern Chinese women. Hum Genet 2013;132(3):301–312. doi:10.1007/s00439-012-1245-4, PMID:23180271.
[29] Zhao X, Yang L, Hu J. Down-regulation of miR-27a might inhibit proliferation and drug resistance of gastric cancer cells. J Exp Clin Cancer Res 2011;30(1):55. doi:10.1186/1756-9966-30-55, PMID:21569481.
[30] Duwe L, Munoz-Garrido P, Lewinska M, Lafuente-Barquero J, Satriano L, Høgdall D, *et al*. MicroRNA-27a-3p targets FoxO signalling to induce tumour-like phenotypes in bile duct cells. J Hepatol 2023;78(2):364–375. doi:10.1016/j.jhep.2022.10.012, PMID:36848245.
[31] Watson JA, Bryan K, Williams R, Popov S, Vujanic G, Coulomb A, *et al*. miRNA profiles as a predictor of chemoresponsiveness in Wilms' tumor blastema. PLoS One 2013;8(1):e53417. doi:10.1371/journal.pone.0053417, PMID:23308219.
[32] Guo ZT, Yu Q, Miao C, Ge W, Li P. MicroRNA-27a-5p Inhibits Proliferation, Migration, and Invasion and Promotes Apoptosis of Wilms' Tumor Cell by Targeting PBOV1. Mol Cell Biol 2022;42(8):e0039721. doi:10.1128/mcb.00397-21, PMID:35862727.
[33] Li Y, Li J, Sun X, Chen J, Sun X, Zheng J, *et al*. MicroRNA-27a functions as a tumor suppressor in renal cell carcinoma by targeting epidermal growth factor receptor. Oncol Lett 2016;11(6):4217–4223. doi:10.3892/ol.2016.4500, PMID:27313769.
[34] Sun Y, Yang X, Liu M, Tang H. Corrigendum to "B4GALT3 up-regulation by miR-27a contributes to the oncogenic activity in human cervical cancer cells." [Canc. Lett. 375 (2016) 284-292]. Cancer Lett 2020;493:16–18. doi:10.1016/j.canlet.2020.07.019, PMID:32823018.
[35] Colangelo T, Polcaro G, Ziccardi P, Pucci B, Muccillo L, Galgani M, *et al*. Proteomic screening identifies calreticulin as a miR-27a direct target repressing MHC class I cell surface exposure in colorectal cancer. Cell Death Dis 2016;7(2):e2120. doi:10.1038/cddis.2016.28, PMID:26913609.
[36] Xu Q, Chen TJ, He CY, Sun LP, Liu JW, Yuan Y. MiR-27a rs895819 is involved in increased atrophic gastritis risk, improved gastric cancer prognosis and negative interaction with Helicobacter pylori. Sci Rep 2017;7:41307. doi:10.1038/srep41307, PMID:28150722.
[37] Shi TY, He J, Qiu LX, Zhu ML, Wang MY, Zhou XY, *et al*. Association between XPF polymorphisms and cancer risk: a meta-analysis. PLoS One 2012;7(7):e38606. doi:10.1371/journal.pone.0038606, PMID:22768293.