Abstract. Long noncoding RNA (lncRNA) has an important role in regulating non-small cell lung cancer (NSCLC) progression. The present study aimed to investigate the effect of LINC00210 in NSCLC progression in order to provide a novel treatment target for patients with NSCLC. A total of 39 NSCLC patients were obtained and divided into LINC00210 high expression and low expression groups. Subsequently, the 5-year survival rate from this patient cohort was analyzed. The SK-MES-1 and A549 NSCLC and the human 16-HBE bronchial epithelial cell lines were utilized to investigate expression level of LIN00210. A549 cells were used to investigate cell proliferation, migration and invasive abilities using Cell Counting kit 8, Transwell and Matrigel assays, respectively. In addition, the luciferase reporter gene assay was performed to investigate the potential target of LINC00210. Reverse transcription-quantitative PCR was used to determine LINC00210 and microRNA (miR)-328-5p expression levels in NSCLC tissues and tumor cell lines (SK-MES-1 and A549). The results demonstrated that LINC00210 was upregulated in NSCLC tissues and cell lines compared with that in normal tissues and 16-HBE cells, and that LINC00210 expression was associated with a poor prognosis in patients with NSCLC (P<0.05). Furthermore, A549 cell transfection with small interfering (si)LINC00210#1 and siLINC00210#2 induced a significant decrease in cell proliferation, and migratory and invasive ability compared with that in the control groups (P<0.05). In addition, miR-328-5p overexpression was stimulated by knockdown of LINC00210. Furthermore, A549 cells transfected with siLINC00210#1 and miR-328-5p inhibitor exhibited a significant increase in cell proliferation, and migratory and invasive ability compared with that in A549 cells transfected with siLINC00210#1. These findings suggest that LINC00210 may serve as an oncogenic role in NSCLC by sponging miR-328-5p.

LINC00210 plays oncogenic roles in non-small cell lung cancer by sponging microRNA-328-5p

ZHENGJIA LIU1*, LEI XU1*, KEJIAN ZHANG2, BO GUO1, ZHI CUI1 and NAN GAO1

1Department of Thoracic Surgery, China-Japan Union Hospital of Jilin University, Changchun, Jilin 130033; 2Department of Thoracic Surgery, Jilin Cancer Hospital, Changchun, Jilin 130021, P.R. China

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Correspondence to: Dr Nan Gao, Department of Thoracic Surgery, China-Japan Union Hospital of Jilin University, 126 XianTai Road, Changchun, Jilin 130033, P.R. China
E-mail: nangaoaa@163.com

*Contributed equally

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induce apoptosis of NSCLC cells by sponge-like adsorption of microRNA-195 therefore, increasing sensitivity to radiotherapy of these cells (8). LncRNAs may therefore be used to develop novel targeted therapy to treat NSCLC.

The present study determined the expression of LINC00210 in NSCLC tumor tissues and cells and investigated its effects on NSCLC progression. Moreover, a previous study identified that LINC00210 sponges miR-328-5p (9). Thus, this present study explored whether LINC00210 also sponges miR-328-5p in NSCLC. Through luciferase reporter assays, it was demonstrated that LINC00210 targeted miR-328-5p to promote NSCLC progression. The findings from the present study may provide a novel therapeutic target for the diagnosis and treatment of NSCLC.

Materials and methods

Ethical statement. The present study was approved by the Ethics Committee of China-Japan Union Hospital of Jilin University and patients provided written informed consent.

Patients and samples. A total of 39 patients who were diagnosed for the first time with NSCLC, according to the grading system of the American Joint Committee on Cancer (10), at the China-Japan Union Hospital of Jilin University, between March 2010 and July 2012, were included in the present study. Patients treated with radiotherapy or chemotherapy before surgery were excluded. The 39 patients with NSCLC included 29 men and 10 females, and their mean age was 58.7±9.2 years. The clinicopathological characteristics of all patients are presented in Table I. Patients' carcinoma tissues and corresponding adjacent normal tissues (at least 3 cm from tumor tissues) were collected during resection and immediately stored at -20°C. Following surgery, all patients were followed-up every 3 months for 5 years through telephone consultations, to analyze their 5-year survival rate using Kaplan-Meier survival analysis.

Cell culture. The SK-MES-1 and A549 NSCLC, and the human 16-HBE bronchial epithelial cell line were all provided by The Cell Bank of Type Culture Collection of the Chinese Academy of Sciences. All cells were maintained separately in Dulbecco's Modified Eagle medium (DMEM) supplemented with 10% FBS (Invitrogen; Thermo Fisher Scientific, Inc.), 100 μg/ml streptomycin and 100 U/ml penicillin and cultured at 37°C in a humidified incubator containing 5% CO2.

Transfection. A total of two siRNAs against LINC00210 (siRNA#1: 5'-GGUCUCAUCUCAUCUAAUU-3' and siRNA#2: 5'-CGGUAUUAUGACCCACUACUUU-3') as well as LINC00210 scramble siRNA negative control (NC) (5'-AAUCUCCGAAGCUGUACGG-3'), miR-328-5p mimics (5'-GGGGGGGCGAGGAGGGCGUAGGG-3'), miR-328-5p NC (scrambled; 5'-UCACACUGCUUGAAGAZAGA-3') and miR-328-5p inhibitors (5'-CCC TGAACCCCCTCTGCCCCCC-3') were all synthesized by Shanghai GenePharma Co., Ltd. A549 cells cultured in serum free DMEM were transfected with 100 nM of the siRNA/miRNA/miRNA inhibitor, using Lipofectamine® 2000 (Thermo Fisher Scientific, Inc.). After transfection, A549 cells were grouped as follows: siLINC00210#1 group (transfected with LINC00210 siRNA#1), siLINC00210#2 group (transfected with LINC00210 siRNA#2), scramble group (transfected with LINC00210 siRNA NC), miR-NC group (transfected with miR-328-5p NC), miR-328-5p group (transfected with miR-328-5p mimics), siLINC00210#1 + miR-328-5p inhibitor group (co-transfected with LINC00210 siRNA#1 and miR-328-5p inhibitors). The transfection efficiency was confirmed using reverse transcription-quantitative PCR (RT-qPCR), 24 h after transfection and transfected cells were used for following experiments.

Cell proliferation assay. Cell proliferation was measured using Cell Counting Kit-8 (CCK8) assay (Sigma-Aldrich; Merck KGaA). Transfected A549 cells were seeded into 96-well plates (2x103 cells/well) and cultured for 24, 48, 72 and 96 h. CCK8 solution (20 μl; 0.5 mg/ml; Sigma-Aldrich; Merck KGaA) was added into each well and incubated for 2 h at 37°C. Absorbance was measured at 450 nm using a micro-plate reader (Thermo Labsystems).

Transwell assay. To investigate cell invasion, 24-well Transwell chambers were pre-coated with Matrigel (BD Biosciences) for 30 min at 37°C and were inserted into 24-well plates containing 1 ml DMEM supplemented with 10% FBS. A total of 100 μl of A549 cells in serum free-suspension (1x103 cells/ml) was added to 24-well Transwell chambers and incubated in a humidified incubator at 37°C with 5% CO2. After 2 days cells that had not penetrated the membrane were removed with a cotton swab. Cells that penetrated and adhered to the lower side of the membrane were fixed with 4% paraformaldehyde for 30 min at room temperature and stained with crystal violet (0.1%) for 10 min at room temperature. The number of invading cells was counted in five random fields under the light microscope at 100x magnification. Cell number was counted using ImageJ (version 1.41; National Institutes of Health). For the assessment of cell migration, the same method was performed, although the 24-well Transwell chambers, were not pre-coated with Matrigel.

Luciferase reporter gene assay. To determine if LINC0020 is a target of miR-328-5p the target gene prediction software miRcode 11 (http://www.mircode.org/) was used. Mutant (Mut) and wild-type (WT) sequences of the LINC00210 containing the 3 untranslated region were designed and amplified using PCR. A549 cells from the miR-NC and miR-328-5p groups were seeded in 24-well plates (1x104 cells per well), and transfected with pGL3-LINC00210-Mut and pGL3-LINC00210-WT plasmids (Promega Corporation), respectively using Lipofectamine® 2000, and cultured for 24 h at 37°C. Cells were collected and firefly and Renilla luciferase activity was detected using a Dual-Luciferase Reporter Assay System kit (Promega Corporation) according to the manufacturer's protocol. Firefly luciferase activity was normalized to Renilla luciferase activity.

RT-qPCR. Total RNA was extracted from tissue samples and cells using TRIzol® reagent (Invitrogen; Thermo Fisher Scientific, Inc.) according to the manufacturer's protocol. Total RNA was reverse transcribed into cDNA at 37°C for 15 min.
Table I. Association between LINC00210 expression and the clinicopathological characteristics of patients with non-small cell lung cancer.

| Characteristics     | LINC00210 low expression (n=18) | LINC00210 high expression (n=21) | P-value  |
|---------------------|---------------------------------|---------------------------------|----------|
| Sex                 |                                  |                                 | 0.6508   |
| Male                | 14                               | 15                              |          |
| Female              | 4                                | 6                               |          |
| Age, years          |                                  |                                 | 0.5189   |
| ≥60                 | 13                               | 17                              |          |
| >60                 | 5                                | 4                               |          |
| Tumor size, cm      |                                  |                                 | 0.0071 a |
| ≤3                  | 12                               | 5                               |          |
| >3                  | 6                                | 16                              |          |
| TNM stage           |                                  |                                 | 0.0160 a |
| I and II            | 9                                | 3                               |          |
| III and IV          | 9                                | 18                              |          |
| Lymph node metastasis |                                |                                 | 0.0174 a |
| Negative            | 12                               | 6                               |          |
| Positive            | 6                                | 15                              |          |

*P<0.05 (χ² test). TNM, Tumor-Node-Metastasis.

LINC00210 expression level and the clinicopathological characteristics of patients (n=18; P<0.05; Fig. 1C; Log Rank χ² value, 4.6). Analysis of the association between LINC00210 expression level and the clinicopathological characteristics of patients with NSCLC revealed that LINC00210 expression level was significantly associated with tumor size, TNM stage and lymph node metastasis (Table I; P<0.05). High LINC00210 expression level may therefore be used to predict larger tumor size, advanced tumor stage and positive lymph node metastasis. The upregulation of LINC00210 in patients with NSCLC indicated a poor prognosis.

LINC00210 knockdown inhibits A549 cell proliferation, and migratory and invasive abilities. A549 cells in the siLINC00210#1 and siLINC00210#2 groups exhibited a significantly decreased LINC00210 expression level compared with that in A549 cells from the scramble NC group (P<0.05; Fig. 2A), indicating that LINC00210 was successfully down-regulated in A549 cells following transfection. The results from the CCK8 assay revealed that the absorbance was significantly reduced in A549 cells in the siLINC00210#1 and siLINC00210#2 groups compared with that in A549 cells from the scramble NC group (P<0.05; Fig. 2B). In addition, transfection with siLINC00210#1 and siLINC00210#2 induced a significant decrease in the migratory and invasive ability of A549 cells compared with that in A549 cells from the scramble NC group (P<0.05; Fig. 2C and D).
LINC00210 promotes miR-328-5p expression. The distribution of LINC00210 expression level in A549 cells was investigated. As presented in Fig. 3A, LINC00210 was mainly distributed in the cytoplasm. A previous study reported that LINC00210 can interact with miR-328-5p (9). Through bioinformatics analysis, miR-328-5p was also identified as a potential target of LINC00210, therefore miR-328-5p was investigated in the present study. The WT and Mut sequences of LINC00210 were designed separately, and their binding sites to miR-328-5p were presented in Fig. 3B. A luciferase reporter assay was performed following A549 cell transfection with miR-328-5p mimics and WT- or Mut-LINC00210 reporter plasmid. The results revealed that miR-328-5p mimics significantly inhibited the relative luciferase activity of WT-LINC00210, but not Mut-LINC00210 (Fig. 3C; P<0.05) compared with that in miR-NC group. Furthermore, A549 cells from the siLINC00210#1 and siLINC00210#2 groups exhibited a significant increase in miR-328-5p expression level compared with that in A549 cells from the scramble NC group (P<0.05; Fig. 3D). The results from the Pearson's correlation analysis revealed a negative correlation between LINC00210 and miR-328-5p expression levels in NSCLC tissues.
These findings suggest that LINC00210 knockdown may directly promote miR-328-5p expression.

LINC00210 knockdown inhibits A549 cell proliferation, and migratory and invasive ability by promoting miR-328-5p expression. miR-328-5p inhibitor significantly inhibited the expression of miR-328-5p (Fig. 4A). For further experiments, siLINC00210#1 was chosen because it produced the largest knockdown. A549 cells from the siLINC00210#1 group exhibited a significant decrease in cell proliferation, and in the migratory and invasive ability compared with that in A549 cells from the scramble NC group and the siLINC00210#1 + miR-328-5p inhibitor group (P<0.05; Fig. 4B-D). However, there was no significant difference in the proliferation, and migratory and invasive ability of A549 cells between the scramble NC group and the siLINC00210#1 + miR-328-5p inhibitor group (Fig. 4B-D). These results suggest that LINC00210 knockdown may inhibit A549 cell proliferation, and migratory and invasive ability by promoting miR-328-5p expression.

Discussion

The present study hypothesized that LINC00210 may be considered as a novel diagnostic tool and treatment target for patients with NSCLC. A high LINC00210 expression level was also found in patients with NSCLC and was associated with poor prognosis. In addition, the results from the present study revealed that LINC00210 was primarily expressed in the cytoplasm, and that knockdown of LINC00210 could inhibit A549 cell proliferation, and migratory and invasive abilities by promoting miR-328-5p expression. LINC00210 may therefore serve an oncogenic role in NSCLC cells by sponging miR-328-5p.

lncRNAs can directly interact with DNA, mRNA or protein to regulate chromatin modification or structure, transcription and translation, therefore regulating numerous physiological and pathological processes, including cell proliferation or differentiation, stem cell reprogramming, tumorigenesis or drug resistance (12). lncRNAs are divided into carcinogenic lncRNAs and tumor suppressor lncRNAs. As with other tumor types, such as colon cancer and liver cancer (6), the upregulation of carcinogenic lncRNAs in NSCLC can enhance cell proliferation as well as the migratory and invasive capacity, and reduce tumor cell apoptosis and tumor drug sensitivity, including MALAT1, HOTAIR and AFAP1 (13-17). Recently, numerous carcinogenic lncRNAs have been discovered in NSCLC, including HOTAIR, MALAT1, colon cancer associated transcript 2, H19 imprinted maternally expressed transcript and AFAP1 antisense RNA 1 (13‑17), whereas SPRY4 intronic transcript 1, maternally expressed 3, GAS6 antisense RNA 1, growth arrest specific 5 and promoter of CDKN1A antisense DNA damage activated RNA PANDAR have been reported as tumor suppressor lncRNAs in NSCLC (18 -22). LINC00210 has rarely been investigated in human diseases. A previous study investigated the effect of LINC00210 in liver cancer, and reported that LINC00210 is overexpressed in liver cancer, promoting liver tumor initiating cell self-renewal and propagation via the activation of the Wnt/β-catenin signaling pathway (23). Furthermore, another previous study reported that LINC00210 regulates nasopharyngeal carcinoma progression via the miR-328-5p/NOTCH3 axis (11). However, the role of LINC00210 in lung cancer remains unknown. The results from the present study demonstrate that LINC00210 was significantly upregulated in NSCLC tissues, and that high LINC00210 expression level was significantly associated with larger tumor size, advanced TNM stage and lymph node metastasis in patients with NSCLC. In addition, reducing LINC00210 expression using siRNA inhibited NSCLC cell proliferation, and migratory and invasive abilities. As the primary cause of cancer-associated mortality worldwide, the underlying mechanisms of NSCLC have not yet been...
fully elucidated and the actual therapeutic options remain unsatisfactory. The discovery of IncRNAs has improved the clinical understanding of NSCLC tumorigenesis and progression, suggesting that these aforementioned IncRNAs may be considered as promising biomarkers for the early diagnosis and treatment of NSCLC. The results from this paper suggest that LINC00210 may be considered as a novel target for the diagnosis and treatment of patients with NSCLC.

miRNAs represent a class of endogenous, non-coding small RNAs found in eukaryotes. They participate in the regulation of various types of human tumor, such as liver cancer and ovarian cancer (24,25). In the present study, LINC00210 knockdown inhibited A549 cell proliferation, and migratory and invasive ability via the promotion of miR-328-5p expression. miR-328 is a type of microRNA that was been reported to be associated with the progression of numerous tumors. For example, Santasusagna et al (25) reported that miR-328 expression level is reduced in colon cancer tissues compared with that in adjacent normal tissues, and that miR-328 can affect colon cancer progression via solute carrier family 2 member 1/solute carrier family 2 member 1 targeting. In nasopharyngeal carcinoma, miR-328 is considered as a potential prognostic and therapeutic marker due to its inhibiting effect on the epithelial-mesenchymal transition of nasopharyngeal carcinoma cells (26). Liu et al (27) demonstrated that low miR-328 expression can predict a poor prognosis in patients with acute myeloid leukemia. Previous studies also reported that low miR-328 expression level is associated with poor survival in patients with high-grade glioma, and that miR-328 can impair glioma cell proliferation and invasive ability. miR-328 was therefore considered as a favorable prognostic marker in glioma (28,29). Furthermore, a previous study reported that miR-328 is reduced in NSCLC, and that miR-328 upregulation can increase NSCLC cell radiosensitivity via the DNA damage and repair signaling pathway (30). In the present study, miR-328-5p expression level was directly reduced following transfection with LINC00210, which may therefore act as a tumor suppressor in NSCLC.

In conclusion, the results from the present study suggest that LINC00210 may be considered as a novel target for the diagnosis and treatment of patients with NSCLC. In addition, high LINC00210 expression predicted poor prognosis in patients with NSCLC. Following LINC00210 knockdown, NSCLC cell proliferation, and migratory and invasive abilities were reduced. Furthermore, the current study demonstrated that LINC00210 may serve oncogenic role in NSCLC by sponging miR-328-5p. At present, research on LINC00210 is still at an early stage, and further investigation on LINC00210 is required to determine the underlying mechanism of NSCLC and to assist with the development of novel treatment options. The present study did not analyze the effect of LINC00210 on cell cycle; however this will be performed in future studies.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Authors' contributions

ZL and LX contributed to the conception and design of the present study. NG analyzed and interpreted the results, and wrote the manuscript. KZ, BG and ZC performed the experiments and analyzed the data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was approved by The Ethics Committee of China-Japan Union Hospital of Jilin University. Written informed consent was provided from all recruited patients.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

References

1. Siegel RL, Miller KD and Jemal A: Cancer statistics, 2017. CA Cancer J Clin 67: 7-30, 2017.
2. Travis WD: The 2015 WHO classification of lung tumors. Der Pathologe 35 (Suppl 2): S188, 2014.
3. Meseure D, Drak Alsibai K, Nicolas A, Bieche I and Morillon A: Long noncoding RNAs as new architects in cancer epigenetics, prognostic biomarkers, and potential therapeutic targets. Biomed Res Int 2015: 332014, 2015.
4. Yao Y, Li J and Wang L: Large intervening non-coding RNA HOTAIR is an indicator of poor prognosis and a therapeutic target in human cancers. Int J Mol Sci 15: 18985-18999, 2014.
5. Liu Z, Sun M, Lu K, Liu J, Zhang M, Wu W, Wei D, Wang Z and Wang R: The long non-coding RNA HOTAIR contributes to cisplatin resistance of human lung adenocarcinoma cells via downregulation of p21 WAF1/CIP1 expression. PLoS One 8: e77293, 2013.
6. Cheng N, Cai W, Ren S, Li X, Wang Q, Pan H, Zhao M, Li J, Zhang Y, Zhao C, et al: Long non-coding RNAUCA1 induces non-TFA90M acquired resistance to EGFR-TKIs by activating the AKT/Erk pathway in EGFR-mutant non-small cell lung cancer. Oncotarget 6: 23582-23593, 2015.v
7. Guo F, Jiao F, Song Z, Li S, Liu B, Yang H, Zhou Q and Li Z: Regulation of MALAT1 expression by TD433 controls the migration and invasion of non-small cell lung cancer cells in vitro. Biochem Biophys Res Commun 465: 293-298, 2015.
8. Wu D, Li Y, Zhang H and Hu X: Knockdown of Lncrna PVT1 enhances radiosensitivity in non-small cell lung cancer by silencing the miR-195-5p. Cell Physiol Biochem 42: 2453-2466, 2017.
9. Zhang S, Li P, Zhao L and Xu L: LINC00210 as a miR-328-5p sponge promotes nasopharyngeal carcinoma tumorigenesis by activating NOTCH3 pathway. Biosci Rep 38: BSR20181168, 2018.
10. Amin MB, Greene FL, Edge SB, Compton CC, Gershenson JD, Brookland RK, Meyer L, Gress DM, Byrd DR and Winchester DP: The eighth edition AJCC Cancer Staging Manual: Continuing to evolve as a novel biomarker for diagnosis and monitoring of non-small cell lung cancer. Technol Cancer Res Treat 16: 1060-1066, 2017.
11. Livak KJ and Schmittgen TD: Analysis of relative gene expression data using real-time quantitative PCR and the 2(-Delta Delta C(T)) method. Methods 25: 402-408, 2001.
12. Geng C, Ziyun W, Dongqing W, Chengxiang Q, Mingli X, Xi L, Qupeng Z, Guoying Y and Qinghua C: LncRNA Disease: A database for long non-coding RNA-associated diseases. Nucleic Acids Res 42: D1983-D1986, 2015.
13. Li N, Wang Y, Liu X, Luo P, Jing W, Zhu M and Tu J: Identification of circulating long noncoding RNA HOTAIR as a novel biomarker for diagnosis and monitoring of non-small cell lung cancer. Technol Cancer Res Treat 16: 1060-1066, 2017.
14. Li S, Mei Z, Hu HB and Zhang X: The IncRNA MALAT1 contributes to non-small cell lung cancer development via modulating miR-124/STAT3 axis. J Cell Physiol 233: 6679-6688, 2018.
15. Zhao Z, Wang J, Wang S, Chang H, Zhang T and Qu J: LncRNA CCAT2 promotes tumorigenesis by over-expressed PAX5 in non-small cell lung cancer. Biomed Pharmaecoher 87: 692-697, 2017.
16. Huang Z, Lei W, Hu HB, Zhang H and Zhu Y: H19 promotes non-small cell lung cancer (NSCLC) development through STAT3 signaling via sponging mir-17. J Cell Physiol 233: 6768-6776, 2018.
17. Deng J, Liang Y, Liu C, He S and Wang S: The up-regulation of long non-coding RNA AFAP1-ASI is associated with the poor prognosis of Patients with NSCLC. Biomed Pharmacother 75: 8-11, 2015.
18. Sun M, Liu XH, Lu KH, Nie FQ, Xia R, Kong R, Yang JS, Xu TP, Liu YW, Zou YF, et al: EZH2-mediated epigenetic suppression of long non-coding RNA SPRY4-IT1 promotes NSCLC cell proliferation and metastasis by affecting the epithelial-mesenchymal transition. Cell Death Dis 5: e2398, 2014.
19. Lu KH, Li W, Liu XH, Sun M, Zhang ML, Wu WQ, Xie WP and Hou YY: Long non-coding RNA MEG3 inhibits NSCLC cells proliferation and induces cell apoptosis by affecting p53 expression. BMC Cancer 13: 461, 2013.
20. Han L, Kong R, Yin DD, Zhang EB, Xu TP, De W and Shu YQ: Low expression of long non-coding RNA GAS6-ASI predicts a poor prognosis in patients with NSCLC. Med Oncol 30: 694, 2013.
21. Mei Y, Si J, Wang Y, Huang Z, Zhu H, Feng S, Wu X and Wu L: Long non-coding RNA GAS5 suppresses tumorigenesis by inhibiting miR-23a 5 expression in non-small cell lung cancer. Oncol Res 25: 1027-1037, 2017.
22. Han L, Zhang E, Yin D, Kong R, Xu T, Chen W, Xia R, Shu Y and De W: Low expression of long non-coding RNA PANDAR predicts a poor prognosis of non-small cell lung cancer and affects cell apoptosis by regulating Bel-2. Cell Death Dis 6: e1665, 2015.
23. Fu X, Zhu X, Qin F, Zhang Y, Lin J, Ding Y, Yang Z, Zhang Y, Wang L, Zhang Q and Gao Q: Linc00210 drives Wnt/b-catenin signaling activation and liver tumor progression through CTNNB1-dependent manner. Mol Cancer 17: 73, 2018.
24. Li J, Li Q, Huang H, Li Y, Li L, Hou W and You Z: Overexpression of miRNA-221 promotes cell proliferation by targeting the apoptotic protease activating factor-1 and indicates a poor prognosis in ovarian cancer. Int J Oncol 50: 1087-1096, 2017.
25. Santasusagna S, Moreno I, Navarro A, Muñoz C, Martinez F, Núñez E, Hernández R, Castellano J and Monzo M: miR-328 mediates a metabolic shift in colon cancer cells by targeting SLC2A1/GLUT1. Clin Transl Oncol 20: 1161-1167, 2018.
26. Lin CH, Chiang MC and Chen YJ: MicroRNA-328 inhibits migration and epithelial-mesenchymal transition by targeting CD44 in nasopharyngeal carcinoma cells. Onco Targets Ther 11: 2375-2385, 2018.
27. Liu L, Chen RA, Zhang Y, Fan W, Xiao F and Yan X: Low expression of circulating microRNA-328 is associated with poor prognosis in patients with acute myeloid leukemia. Diagn Pathol 10: 109, 2015.
28. Yuan J, Zheng Z, Zheng Y, Lu X, Xu L and Lin L: microRNA-328 is a favorable prognostic marker in human glioma: a suppressing invasive and proliferative phenotypes of malignant cells. Int J Neurosci 126: 145-153, 2016.
29. Wu Z, Sun L, Wang H, Yao J, Jiang C, Xu W and Yang Z: MiR-328 expression is decreased in high-grade gliomas and is associated with worse survival in primary glioblastoma. PLoS One 7: e47207, 2012.
30. Ma W, Ma CN, Zhou NN, Li XD and Zhang YJ: Up-regulation of miR-328-3p sensitizes non-small cell lung cancer to radiotherapy. Sci Rep 6: 31651, 2016.