LINC00511 is a newly identified IncRNA that is up-regulated in many types of human cancers and may serve as an oncogenic IncRNA. However, there was no report about the role of LINC00511 in cervical cancer. Therefore, we investigated the clinical value of LINC00511 in cervical cancer patients via analyzing the correlation between LINC00511 expression and clinicopathological features. Moreover, we performed loss-of-function study to estimate the effect of LINC00511 on cervical cancer cell proliferation, migration, and invasion. In our study, we found LINC00511 expression levels were increased in cervical cancer tissues and cell lines compared with adjacent normal tissues and normal cervical epithelial cell line, respectively. High LINC00511 expression was correlated with advanced clinical stage, large tumor size, histological type of adenocarcinoma, and present lymph node metastasis, distant metastasis, and poor overall survival in cervical cancer patients. The in vitro studies indicated that knockdown of LINC00511 inhibited cervical cancer cell proliferation, migration, and invasion. In conclusion, LINC00511 acts as oncogenic IncRNA in cervical cancer, and may be a novel biomarker and potential therapeutic target for cervical cancer patients.

Introduction

Cervical cancer is the fourth most common female malignancy in the world accounting for approximately 6.6% of all newly diagnosed female cancers [1]. Meanwhile, cervical cancer ranks the fourth leading cause of cancer-related death in women worldwide accounting for 311365 deaths in 2018 [1]. Cervical cancer often derives from abnormal cell growth with human papillomavirus (HPV) infection on the cervix [2]. Recent decades, the generalization of disease screening and introduction of HPV vaccines jointly result in a decreasing incidence trend for cervical cancer in most countries [3,4]. Regrettably, the improvement of cervical cancer treatment strategy is limited [5,6]. Surgery, chemotherapy, and radiotherapy are still the major therapies for cervical cancer patients [7–9]. Therefore, it is necessary to investigate the molecular mechanisms of cervical cancer development for gaining novel therapeutic targets.

Long noncoding RNAs (lncRNAs) are considered a type of non-coding RNAs consisting of more than 200 nucleotides without coding protein potential [10]. More and more researches suggested lncRNAs are involved in human cancer development by regulating tumor cell proliferation, apoptosis, cell-cycle, migration, invasion, and autophagy [11,12]. Long intergenic noncoding RNA 00511 (LINC00511, also known as onco-lncRNA-12), is a newly identified carcinogenic IncRNA that maps to chromosome 17q24.3 [13]. Up to now, LINC00511 has been showed to be overexpressed in many kinds of tumor tissues, and exert oncogenic effects on lung cancer [14,15], breast cancer [16–21], pancreatic cancer [22], bladder cancer [23], osteosarcoma [24], and tongue squamous cell carcinoma [25]. In preliminary experiment, we found levels of LINC00511 expression was also increased in cervical cancer compared with adjacent...
normal tissues. Then, we guessed that LINC00511 functions as oncogenic lncRNA in cervical cancer. Therefore, we investigated the clinical value of LINC00511 in cervical cancer patients via analyzing the correlation between LINC00511 expression and clinicopathological features. Moreover, we performed loss-of-function study to estimate the effect of LINC00511 on cervical cancer cell proliferation, migration, and invasion.

**Materials and methods**

**Clinical samples**

Total 92 cervical cancer tissues and 40 adjacent normal tissues were obtained from volunteer patients with cervical cancer who underwent surgery or biopsy at Daqing Oilfield General Hospital, Rizhao Central Hospital or The Affiliated Hospital of Qingdao University. All clinical tissue samples were promptly frozen in liquid nitrogen and maintained at −80°C until RNA extraction. No radiotherapy and chemotherapy were performed before surgery or biopsy. The study procedures were reviewed and approved by the Ethics Committee of Daqing Oilfield General Hospital, Rizhao Central Hospital and The Affiliated Hospital of Qingdao University. All patients signed and provided written informed consents.

**Quantitative real time PCR**

Total RNA was extracted from tissues and cells by using Trizol reagents (Invitrogen, Carlsbad, CA, U.S.A.), and transcribed into complementary DNA (cDNA) using PrimeScript RT Master Mix (Takara Biomedical Technology, Beijing, China). Then, TB Green Premix Ex Taq II (Takara Biomedical Technology, Beijing, China) was used to conducted quantitative real time PCR (qRT-PCR) at ABI 7500 PCR System (Applied Biosystems, Foster City, CA, U.S.A.) according to the instructions. The sequences of primers used in the present study were LINC00511, 5′-CGCAAGGACCCCTGGTACC-3′ (forward) and 5′-GAAGGCGGATCGTCTCTCAG-3′ (reverse); GAPDH, 5′-GTTGTCTCTGGACTTCACAACA-3′ (forward) and 5′-GTTGCTGTAGCCAAATTCTGTTG-3′ (reverse). The relative expression was standardized by GAPDH.

**Cell culture**

Human cervical cancer cell lines (SiHa, HeLa, C33A, and Caski) and normal cervical epithelial cell line (Ect1/E6E7) were maintained in Roswell Park Memorial Institute (RPMI)-1640 medium with 10% fetal bovine serum (FBS) in a humidified chamber with 5% CO₂ at 37°C.

**Cell transfection**

For down-regulation of LINC00511, siRNA-LINC00511 (5′-CCCAUGUCCUGUCUGCCUUUGACU-3′) and siRNA-NC were synthesized by Shanghai Invitrogen Biotechnology Co., Ltd. Cell transfection was performed by using Lipofectamine 3000 reagents (Invitrogen, Carlsbad, CA, U.S.A.) according to manufacturer’s instructions.

**Cell Counting Kit-8 assay**

Cell proliferation was estimated by Cell Counting Kit-8 (CCK-8) assay (Dojindo Molecular Technologies, Kumamoto, Japan). Transfected cervical cancer cells were (5 × 10⁵ per well) were seeded in the 96-well microtiter plates. At 24, 48, 72, and 96 h, 10 μl CCK-8 solution was added into each well, and the cells were sequentially cultured at 37°C for 2 h. The absorbance at 450 nm was detected using a microplate reader (Thermo Fisher Scientific, Waltham, MA, U.S.A.).

**Cell migration and invasion assays**

For cell invasion assay, transwell chamber with Matrigel Matrix (BD Biosciences, Franklin Lakes, NJ, U.S.A.). Transfected cervical cancer cells were seeded in the 96-well microtiter plates. At 24, 48, 72, and 96 h, 10 μl CCK-8 solution was added into each well, and the cells were sequentially cultured at 37°C for 2 h. The absorbance at 450 nm was detected using a microplate reader (Thermo Fisher Scientific, Waltham, MA, U.S.A.).

**Cell migration and invasion assays**

For cell invasion assay, transwell chamber with Matrigel Matrix (BD Biosciences, Franklin Lakes, NJ, U.S.A.). For cell migration assay, there was no Matrigel Matrix in transwell chamber. Briefly, 600 μl RPMI-1640 medium containing with 10% FBS was added into lower chamber, and 100 μl serum-free RPMI-1640 medium with 5 × 10⁴ cells was added into the upper chamber. After 24 h culture under 37°C, cells located at upper chamber were cleaned out, and cells at lower chamber were fixed with methanol and stained with Giemsa solution. Then, a light microscope was used to count these stained cells with five randomly selected areas.

**Statistical analysis**

SPSS 17.0 software (SPSS, Chicago, IL, U.S.A.) was applied for statistical analysis. All experiments were performed triplicate independently. The paired t-test was used for comparisons of the differential LINC00511 expression in cervical cancer tissues and adjacent normal tissues. The Student’ t-test was used to estimate the significance of differences.
Figure 1. LINC00511 is high-expressed in cervical cancer tissues and cell lines
(A) LINC00511 expression was significantly higher in cervical cancer tissues than in adjacent normal tissues. (B) LINC00511 expression was higher in cervical cancer cell lines (SiHa, HeLa, C33A, and Caski) than normal cervical epithelial cell line.

Results
LINC00511 is high-expressed in cervical cancer tissues and cell lines
We first examined LINC00511 expression in 40 pairs of cervical cancer tissues and adjacent normal tissues using qRT-PCR, and found LINC00511 expression was significantly higher in cervical cancer tissues than in adjacent normal tissues (Figure 1A). Subsequently, the qRT-PCR assay also confirmed the high-expression of LINC00511 in cervical cancer cell lines (SiHa, HeLa, C33A, and Caski) as compared with normal cervical epithelial cell line (Ect1/E6E7) (Figure 1B).

High LINC00511 expression is correlated with clinical progression in cervical cancer patients
To assess the clinical significance of LINC00511 in cervical cancer patients, we studied the correlations between LINC00511 expression and clinicopathologic parameters. All cervical cancer patients were subclassified into high LINC00511 expression group (n=46) and low LINC00511 expression group (n=46). As shown in Table 1, we observed that high LINC00511 expression was correlated with advanced clinical stage, large tumor size, histological type of adenocarcinoma, and present lymph node metastasis and distant metastasis in cervical cancer patients. However LINC00511 expression had no correction with age and HPV infection (Table 1).

High LINC00511 expression is correlated with unfavorable prognosis in cervical cancer patients
To further demonstrate the potential prognostic value of LINC00511, we explored the prognostic impact of LINC00511 on overall survival in cervical cancer patients. We analyzed the relationship between LINC00511 expression and overall survival of cervical cancer patients, and found patients in high LINC00511 expression group had obviously short overall survival compared with those in low LINC00511 expression group (Figure 2). Furthermore, the univariate Cox regression model indicated clinical stage, tumor size, lymph node metastasis, distant metastasis, histological type, and LINC00511 expression were identified as prognostic factors for overall survival in cervical cancer patients (Table 2). Meanwhile, high LINC00511 expression was showed to be an independent poor prognostic factor in multivariate Cox regression model (Table 2).

LINC00511 acts as a tumor promoter by enhancing cell proliferation, migration, and invasion in cervical cancer
To evaluate the biological functions of LINC00511 during cervical cancer progression, we conducted loss-of-function study in cervical cancer cells. Then, HeLa and C33A cells were chosen for following studies in vitro due to relative high expression of LINC00511. First, HeLa and C33A cells were transfected with siRNA-LINC00511 to reduce LINC00511 expression (Figure 3A). The results of CCK-8 suggested knockdown of LINC00511 obviously suppressed cell prolif-
Figure 2. High LINC00511 expression is correlated with unfavorable prognosis in cervical cancer patients
Cervical cancer patients in high LINC00511 expression group had obviously short overall survival compared with those in low LINC00511 expression group.

Figure 3. LINC00511 acts as a tumor promoter by enhancing cell proliferation, migration, and invasion in cervical cancer
(A) HeLa and C33A cells were transfected with siRNA-LINC00511 to reduce LINC00511 expression. (B) Knockdown of obviously suppressed cell proliferation ability of HeLa and C33A cells. (C) Knockdown of LINC00511 inhibited cell migration ability of HeLa and C33A cells. (D) Knockdown of LINC00511 depressed cell invasion ability of HeLa and C33A cells. Each experiment was independently performed in triplicate. *P<0.001.
Table 1 Relationships between LINC00511 expression and clinicopathological characteristics in cervical cancer

| Characteristics                  | n   | LINC00511       |     |     |     |
|----------------------------------|-----|----------------|-----|-----|-----|
|                                  |     | High expression| Low expression| p   |
| Age (years)                      |     |                |                |     |
| ≤50                              | 41  | 24             | 17             | 0.142 |
| >50                              | 51  | 22             | 29             |     |
| Clinical stage                   |     |                |                |     |
| I–IIA                            | 38  | 10             | 28             | <0.001 |
| IIIB–IV                          | 54  | 36             | 18             |     |
| Tumor size (cm)                  |     |                |                |     |
| ≤4                               | 40  | 11             | 29             | <0.001 |
| >4                               | 52  | 35             | 17             |     |
| Lymph node metastasis            |     |                |                |     |
| Absent                           | 53  | 16             | 37             | <0.001 |
| Present                          | 39  | 30             | 9              |     |
| Distant metastasis               |     |                |                |     |
| Absent                           | 85  | 39             | 46             | 0.018 |
| Present                          | 7   | 7              | 0              |     |
| Histological type                |     |                |                |     |
| Adenocarcinoma                   | 14  | 13             | 1              | <0.001 |
| Squamous cell carcinoma          | 78  | 33             | 45             |     |
| Histological grade               |     |                |                |     |
| Well                             | 54  | 24             | 30             | 0.204 |
| Moderately/poorly                | 38  | 22             | 16             |     |

Table 2 Summary of univariate and multivariate Cox regression analysis of overall survival in cervical cancer

| Parameter                              | Univariate analysis | Multivariate analysis |
|----------------------------------------|---------------------|-----------------------|
|                                       | P       | HR      | 95% CI    | P       | HR      | 95% CI    |
| Age (year)                             |         |         |           |         |         |           |
| (≤50 vs. >50)                          | 0.429   | 1.252   | 0.717–2.185 |         |         |           |
| Clinical stage                         |         |         |           |         |         |           |
| (I–IIA vs. IIIB–IV)                    | 0.010   | 2.109   | 1.194–3.724 | 0.976   | 1.016   | 0.365–2.829 |
| Tumor size (cm)                        |         |         |           |         |         |           |
| (≤4 vs. >4)                            | 0.002   | 2.459   | 1.386–4.362 | 0.285   | 1.462   | 0.729–2.932 |
| Lymph node metastasis                  |         |         |           |         |         |           |
| (Absent vs. present)                   | <0.001  | 3.080   | 1.724–5.504 | 0.591   | 1.356   | 0.447–4.117 |
| Distant metastasis                     |         |         |           |         |         |           |
| (Absent vs. present)                   | 0.013   | 3.056   | 1.266–7.378 | 0.199   | 0.428   | 0.117–1.561 |
| Histological type                      |         |         |           |         |         |           |
| (Adenocarcinoma vs. squamous cell carcinoma) | <0.001  | 0.228   | 0.108–0.481 | 0.057   | 0.329   | 0.105–1.033 |
| Histological grade                     |         |         |           |         |         |           |
| (Well vs. moderately/poorly)           | 0.872   | 1.046   | 0.605–1.810 |         |         |           |
| LINC00511 expression                   |         |         |           |         |         |           |
| (Low vs. high)                         | <0.001  | 4.100   | 2.309–7.278 | 0.003   | 2.895   | 1.446–5.797 |

HR, hazard ratio; 95% CI, 95% confidence interval.

...eration ability of HeLa and C33A cells (Figure 3B). Moreover, cell migration and invasion assays revealed knockdown of LINC00511 also markedly inhibited cell migration and invasion abilities of HeLa and C33A cells (Figure 3C,D).
Discussion

LINC00511, also known as onco-lncRNA-12, is a newly identified IncRNA that is up-regulated in many types of human cancers and may serve as an oncogenic IncRNA. Initially, Cabanski et al. conducted a pan-cancer analysis of IncRNAs comparing cancer tissue samples and matched normal tissue samples expression levels using RNA-Seq data in eight types of human cancers, and found levels of LINC00511 expression were significantly elevated in invasive breast cancer, lung adenocarcinoma, lung squamous cell carcinoma, colorectal cancer compared with corresponding normal tissues [13]. Subsequently, high expression of LINC00511 was further confirmed in lung adenocarcinoma [14], lung squamous cell carcinoma [14], breast cancer [21], pancreatic cancer [22], bladder cancer [23], osteosarcoma [24], and tongue squamous cell carcinoma [25]. However, the expression pattern of LINC00511 in cervical cancer was still unknown. Thus, we observed the LINC00511 expression in The Cancer Genome Atlas (TCGA) and The Genotype-Tissue Expression (GTEX) databases, and found LINC00511 expression was significantly overexpressed in cervical cancer tissues compared with normal tissues. Furthermore, we further confirmed the LINC00511 expression in cervical cancer tissues and cell lines through qRT-PCR, and found LINC00511 expression levels were increased in cervical cancer tissues and cell lines compared with adjacent normal tissues and normal cervical epithelial cell line, respectively. In addition, we explored the clinical significance of LINC00511 in cervical cancer patients through studying the correlations between LINC00511 expression and clinicopathologic parameters, and found high LINC00511 expression was correlated with advanced clinical stage, large tumor size, histological type of adenocarcinoma, and present lymph node metastasis and distant metastasis. Similarly, Sun et al. suggested LINC00511 overexpression was associated with large tumor size, advanced TNM stage, positive lymph node metastasis and smoking in non-small cell lung cancer patients [15]. Besides, Zhao et al. reported that high LINC00511 expression was correlated with high N stage and early recurrence in patients with pancreatic ductal adenocarcinoma [22]. In breast cancer patients, Lu et al. showed there were positive correlations between LINC00511 expression and clinicopathological parameters including TNM stages, tumor size, lymph node metastasis and distant metastases [21]. However, Ding et al. indicated LINC00511 expression had no correlation with any clinicopathological characteristics in patients with tongue squamous cell carcinoma, which may be due to small sample size [25]. Generally, LINC00511 expression was overexpressed in most types of human cancers, but more studies are necessary to explore the clinical significance of LINC00511 expression in various kinds of cancer.

The prognostic significance of LINC00511 was reported in lung cancer [15], breast cancer [18,21], and pancreatic cancer [22]. In lung cancer patients, Sun et al. found LINC00511 overexpression was correlated with short overall survival, and acted as an independent unfavorable predictor for overall survival [15]. Besides, Xu et al. and Lu et al. congruously showed breast cancer patients with high-expression of LINC00511 had poorer overall survival than patients with low-expression of LINC00511 [18,21]. Zhao et al. demonstrated that high LINC00511 expression predicted poor progression-free survival and overall survival, and served as independent prognostic indicator for overall survival of pancreatic ductal adenocarcinoma patients [22]. In our study, we also found cervical cancer patients in high LINC00511 expression group had obviously short overall survival compared with those in low LINC00511 expression group, and high LINC00511 expression was showed to be an independent poor prognostic factor for overall survival in cervical cancer patients, which was consist with the prognostic value of LINC00511 in other types of human cancer.

LINC00511 exerts oncogenic effects on cell proliferation, cell-cycle, cell apoptosis, migration, invasion, and stemness in human cancers. There was no report about the biological function of LINC00511 in cervical cancer cells. In our study, we preliminarily investigated the effect of LINC00511 on cervical cancer cell proliferation, migration and invasion. We found knockdown of LINC00511 markedly inhibited cervical cancer cell proliferation, migration and invasion. The limitation of our study is lack of the molecular mechanism of LINC00511 in cervical cancer cells. The microRNA and its target were important regulatory mechanism for LINC00511, such as miR-185-3p/E2F1 in breast cancer [21], miR29b-3p/VEGFA in pancreatic cancer [22], miR-15a-3p/Wnt signaling pathway in bladder cancer [23], miR-765/APE1 in osteosarcoma [24] and miR-765/LAMC2 in tongue cancer [25].

In conclusion, LINC00511 expression is increased in cervical cancer tissues and cell lines. High LINC00511 expression is correlated with clinical progression and poor prognosis in cervical cancer patients. Knockdown of LINC00511 inhibits cervical cancer cell proliferation, migration, and invasion.

Author Contribution

Chun-Ling Yu and Fang Yuan designed the experiment, interpreted the data, and prepared the manuscript. Chun-Ling Yu and Xiao-Ling Xu conducted the experiment and analyzed the data.
Competing Interests
The authors declare that there are no competing interests associated with the manuscript.

Funding
This work was supported by the Nature Science Foundation of Shandong [grant number 20160081].

Abbreviations
CCK-8, Cell Counting Kit-8; HPV, human papillomavirus; IncRNA, long noncoding RNA; qRT-PCR, quantitative real time PCR; RPMI, Roswell Park Memorial Institute.

References
1 Bray, F., Ferlay, J., Soerjomataram, I., Siegel, R.L., Torre, L.A. and Jemal, A. (2018) Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J. Clin. 68, 394–424, https://doi.org/10.3322/caac.21492
2 Hillemanns, P., Soergel, P., Hertel, H. and Jentschke, M. (2016) Epidemiology and early detection of cervical cancer. Oncol. Res. Treat. 39, 501–506, https://doi.org/10.1159/000448385
3 Cohen, P.A., Jingran, A., Okkin, A. and Denny, L. (2019) Cervical cancer. Lancet 393, 169–182, https://doi.org/10.1016/S0140-6736(18)32470-X
4 Hu, Z. and Ma, D. (2018) The precision prevention and therapy of HPV-related cervical cancer: new concepts and clinical implications. Cancer Med. 7, 5217–5236, https://doi.org/10.1002/cam4.1501
5 Nahand, J.S., Taghizadeh-Boroujeni, S., Karimzadeh, M., Borran, S., Pourhanifeh, M.H., Moghoofei, M. et al. (2019) microRNAs: new prognostic, diagnostic, and therapeutic biomarkers in cervical cancer. J. Cell. Physiol., https://doi.org/10.1002/jcp.28457
6 Johnson, C.A., James, D., Marzan, A. and Armaso, M. (2019) Cervical cancer: an overview of pathophysiology and management. Semin. Oncol. Nurs., https://doi.org/10.1016/j.socn.2019.02.003
7 Marshall, C., Rajdev, M.A., Somarouthu, B., Ramaiya, N.H. and Alessandrino, F. (2019) Overview of systemic treatment in recurrent and advanced cervical cancer: a primer for radiotherapists. Abdom. Radiol. (NY) 44, 1506–1519, https://doi.org/10.1186/s00261-018-1797-4
8 Angeles, M.A., Martinez-Gomez, C., Migliorelli, F., Voglimacci, M., Figurelli, J., Motton, S. et al. (2018) Novel surgical strategies in the treatment of gynecological malignancies. Curr. Treat. Options Oncol. 19, 73, https://doi.org/10.1007/s11864-018-0582-5
9 Fokom Domguy, J. and Schneller, K.M. (2019) Conservative management of cervical cancer: current status and obstetrical implications. Best Pract. Res. Clin. Obstet. Gynaecol. 55, 79–92, https://doi.org/10.1016/j.bpobgyn.2018.06.009
10 Aalijahan, H. and Ghobadian, S. (2019) Long non-coding RNAs and cervical cancer. Exp. Mol. Pathol. 106, 7–16, https://doi.org/10.1016/j.xmop.2018.10.010
11 Sarfi, M., Abbastabar, M. and Khalili, E. (2019) Long noncoding RNAs biomarker-based cancer assessment. J. Cell. Physiol., https://doi.org/10.1002/jcp.28417
12 Liu, Z., Dai, J. and Shen, H. (2018) Dataset for regulation between IncRNAs and their nearby protein-coding genes in human cancers. Data Brief 19, 1902–1906, https://doi.org/10.1016/j.dib.2018.06.048
13 Cabanski, C.R., White, N.M., Dang, H.X., Silva-Fisher, J.M., Rauck, C.E., Cicka, D. et al. (2015) Pan-cancer transcriptome analysis reveals long noncoding RNAs with conserved function. RNA Biol. 12, 628–642, https://doi.org/10.1080/15476286.2015.1038012
14 Wei, Y. and Zhang, X. (2016) Transcriptome analysis of distinct long non-coding RNA transcriptional fingerprints in lung adenocarcinoma and squamous cell carcinoma. Tumour Biol. 37, 16275–16285, https://doi.org/10.1007/s13277-016-5422-2
15 Sun, C.C., Li, S.J., Li, G., Hua, R.X., Zhou, X.H. and Li, D.J. (2016) Long intergenic noncoding RNA 00511 acts as an oncogene in non-small-cell lung cancer by binding to EZH2 and suppressing p57. Mol. Ther. Nucleic Acids 5, e385, https://doi.org/10.1038/mtna.2016.94
16 Yang, F., Lyu, S., Dong, S., Liu, Y., Zhang, X. and Wang, O. (2016) Expression profile analysis of long noncoding RNA in HER-2-enriched subtype breast cancer by next-generation sequencing and bioinformatics. Onco. Targets Ther. 9, 761–772, https://doi.org/10.15407/OTT9.7664
17 Oh, T.G., Wang, S.M., Acharya, B.R., Goode, J.M., Graham, J.D., Clarke, C.L. et al. (2016) The nuclear receptor, RORgamma, regulates pathways necessary for breast cancer metastasis. EBioMedicine 19, 59–72, https://doi.org/10.1016/j.ebiom.2016.02.028
18 Xu, S., Kong, D., Chen, Q., Ping, Y. and Pang, D. (2017) Oncogenic long noncoding RNA landscape in breast cancer. Mol. Cancer 16, 129, https://doi.org/10.1186/s12943-017-0696-6
19 Xiao, B., Zhang, W., Chen, L., Hang, J., Wang, L., Zhang, R. et al. (2018) Analysis of the miRNA-mRNA-IncRNA network in human estrogen receptor-positive and estrogen receptor-negative breast cancer based on TCGA data. Gene 658, 28–35, https://doi.org/10.1016/j.gene.2018.03.011
20 Kholghi Oskoeei, V., Geranpayeh, L., Omrani, M.D. and Ghafouri-Fard, S. (2018) Assessment of functional variants and expression of long noncoding RNAs in vitamin D receptor signaling in breast cancer. Cancer Manag. Res. 10, 3451–3462, https://doi.org/10.2147/CMAR.S174244
21 Lu, G., Li, Y., Ma, Y., Lu, J., Chen, Y., Jiang, Q. et al. (2018) Long noncoding RNA LINC00511 contributes to breast cancer tumorigenesis and stemness by inducing the miR-185-3p/E2F1/Nanog axis. J. Exp. Clin. Cancer Res. 37, 289, https://doi.org/10.1186/s13046-018-0945-6
22 Zhao, X., Liu, Y., Li, Z., Zheng, S., Wang, L., Li, W. et al. (2018) LINC00511 acts as a competing endogenous RNA to regulate VEGFA expression through sponging hsa-miR-29b-3p in pancreatic ductal adenocarcinoma. J. Cell. Mol. Med. 22, 655–667, https://doi.org/10.1111/jcmm.13351
23 Li, J., Li, Y., Meng, F., Fu, L. and Kong, C. (2018) Knockdown of long non-coding RNA linc00511 suppresses proliferation and promotes apoptosis of bladder cancer cells via suppressing Wnt/beta-catenin signaling pathway. Biosci. Rep. 38
24 Yan, L., Wu, X., Liu, Y. and Xiao, W. (2019) LncRNA Linc00511 promotes osteosarcoma cell proliferation and migration through sponging miR-765. J. Cell. Biochem. 120, 7248–7256

© 2019 The Author(s). This is an open access article published by Portland Press Limited on behalf of the Biochemical Society and distributed under the Creative Commons Attribution License 4.0 (CC BY).
25 Ding, J., Yang, C. and Yang, S. (2018) LINC00511 interacts with miR-765 and modulates tongue squamous cell carcinoma progression by targeting LAMC2. J. Oral Pathol. Med. 47, 468–476, https://doi.org/10.1111/jop.12677