INTRODUCTION

Ovarian cancer (OC) is a common occurred cancer type in female reproductive system (Siegel, Miller, & Jemal, 2018). The 5-year overall survival for OC patients is about 30% due to approximately 75% of cancer patients are diagnosed at late stages (Siegel, Miller, & Jemal, 2017; Sorensen, Schnack, Karlsen, & Hogdall, 2015). Therefore, it is imperative to...
explore the novel targets which can be used for tumor-targeted therapy.

Emerging evidence showed OC carcinogenesis is correlated with aberrantly expressed of long non-coding RNAs (lncRNAs) (Wang, Lu, & Chen, 2019). Even though lncRNA lacks the ability to code proteins, it was reported to regulate various cellular processes and tumorigenesis (Bartonicek, Maag, & Dinger, 2016). For instance, lncRNA FLVCR1 antisense RNA 1 (FLVCR1-AS1) was reported to overexpressed in OC tissues, serum, and cells (Yan et al., 2019). The overexpression of FLVCR1-AS1 was revealed to promote cancer proliferation, metastasis, and epithelial to mesenchymal transition via regulating Yes associated protein 1 (606608) expression via modulating MICRORNA-513, while the knockdown of FLVCR1-AS1 caused opposite effects on OC cell behaviors (Yan et al., 2019). Moreover, lncRNA growth arrest specific 5 (GAS5, 608280) was identified to be elevated expression in epithelial ovarian cancer tissues and cells (Long, Xiong, et al., 2019). Mechanism study showed that GAS5 could recruit transcription factor enhancer of zeste 2 polycym repressive complex 2 subunit (601573) to regulate MAP kinase signal pathway (Long, Song, et al., 2019). Moreover, LncRNA growth arrest specific 5 was found could regulate the response of cancer cells to chemo-reagents. For example, KCNQ1OT1 was found overexpressed in methotrexate resistant colorectal cancer cells (Xian & Zhao, 2019). Also, they showed KCNQ1OT1 was able to affect the chemosensitivity of colorectal cancer cell via targeting protein phosphatase 1 regulatory inhibitor subunit 1B (604399) through sponging MIR-760 (Xian & Zhao, 2019). In colon cancer, KCNQ1OT1 was identified to be elevated expression in colon and rectal adenocarcinoma (Zhang, Yan, Yi, Rui, & Hu, 2019). Also, high expression in colon and rectal adenocarcinoma (Zhang, Yan, Yi, Rui, & Hu, 2019). Moreover, KCNQ1OT1 was revealed to regulate various cellular processes and tumorigenesis (Bartonicek, Maag, & Dinger, 2016). For instance, lncRNA FLVCR1 antisense RNA 1 (FLVCR1-AS1) was reported to overexpressed in OC tissues, serum, and cells (Yan et al., 2019). The overexpression of FLVCR1-AS1 was revealed to promote cancer proliferation, metastasis, and epithelial to mesenchymal transition via regulating Yes associated protein 1 (606608) expression via modulating MICRORNA-513, while the knockdown of FLVCR1-AS1 caused opposite effects on OC cell behaviors (Yan et al., 2019). Moreover, lncRNA growth arrest specific 5 (GAS5, 608280) was identified to be elevated expression in epithelial ovarian cancer tissues and cells (Long, Xiong, et al., 2019). Mechanism study showed that GAS5 could recruit transcription factor enhancer of zeste 2 polycym repressive complex 2 subunit (601573) to regulate MAP kinase signal pathway (Long, Song, et al., 2019). 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Also, they showed KCNQ1OT1 was able to affect the chemosensitivity of colorectal cancer cell via targeting protein phosphatase 1 regulatory inhibitor subunit 1B (604399) through sponging MIR-760 (Xian & Zhao, 2019). In colon cancer, KCNQ1OT1 was also identified to be elevated expression and correlated with poor overall survival of cancer patients (Li et al., 2019). Moreover, it was found knockdown of KCNQ1OT1 inhibits cell proliferation but promotes cell apoptosis through MIR-34a (611172)/autophagy-related 4B cysteine peptidase (611338) (Li et al., 2019). Besides that, KCNQ1OT1 was shown to regulate the osteosarcoma cell behaviors and its response to cisplatin via regulating Kcnq1/DNA methyltransferase 1 mediated KCNQ1 expression (Li et al., 2019). However, its expression and roles of KCNQ1OT1 in OC remains to be explored.

In this study, expression of KCNQ1OT1 in OC tissues and cell lines was explored. Moreover, a series of loss and gain-of-function experiments were performed to investigate the biological roles of KCNQ1OT1. Moreover, the underlying mechanism of KCNQ1OT1 in regulating OC cell behaviors was explored.

2 MATERIALS AND METHODS

2.1 Cell culture

The Roswell Park Memorial Institute-1640 (RPMI-1640) and fetal bovine serum (FBS) purchased from Invitrogen (Thermo Fisher Scientific, Inc., Waltham, MA, USA) was used to incubate OC cells (SKOV3, OVCAR3) and normal ovarian epithelial cell line (IOSE80). The incubation condition was maintained at 37°C and contained 5% of CO2. All these three cells were obtained from American Type Culture Collection (ATCC).

2.2 Cell transfection

For manipulating the expression of KCNQ1OT1, small-interfering RNA (siRNA) for KCNQ1OT1 (si-KCNQ1OT1, 5'-GGTGAAGATAGTTCTGCTTT-3'), overexpression construct for KCNQ1OT1 (pKCNQ1OT1) or calpain 10 (605286, pCAPN10), and the relative controls (si-R-NC, 5'-ATCTATGATTGGTCTATGG-3' and pcDNA3.1) was synthesized by GenScript. MIR-140-5p mimic (5'-CAGUGGUUUUACCUCUGUGA-3') and the corresponding negative control (miR-NC, 5'-GGU CAUCGGAUUGUACUGUA-3') were designed by GenePharm. Cells were transfected using Lipofectamine 2000 (Invitrogen) until incubated to approximately 80% confluence according to the standard protocols.

2.3 Quantitative real-time PCR (qRT-PCR)

To explore relative gene expression level, qRT-PCR was performed on ABI 7,500 system (Applied Biosystem). RNA from cells was extracted using Trizol reagent (Invitrogen) and then reverse transcribed into complementary DNA using First-Strand cDNA Synthesis kit (Invitrogen). qRT-PCR was performed using SYBR Green Mix (Takara, Dalian, Liaoning, China) with the following procedure: 95°C for 10 min, 40 cycles of 95°C for 10 s, 55°C for 10 s, and 72°C for 30 s. Relative expression level was calculated with the 2−ΔΔCt method. Primers used were as follows: KCNQ1OT1: forward: 5'-GCTGCTGAGTATACTGTCTTT-3'; reverse: 5'-TCAGGTGGACATAGATCAGTG-3'. MIR-142-5p: forward: 5'-GCTGCTGAGTATACTGTCTTT-3'; reverse: 5'-CTCATCTTTGGCTCTATGG-3'; U6 snRNA: forward: 5'-U6 snRNA: forward: 5'-CTCATCTTTGGCTCTATGG-3'.
reverse: 5'-AACGCTTACGAATTT-3'. The procedures were as follows: 95°C for 3 min, 40 cycles of 95°C for 10 s, and 58°C for 30 s.

2.4 | Cell counting kit-8 (CCK-8) assay

Cell proliferation ability was analyzed with CCK-8 (Beyotime, Haimen, Jiangsu, China) assay according to the supplier’s instructions. Cell proliferation rate was analyzed at 0, 1, 2, and 3 days after incubation. At these time points, CCK-8 reagent was added to each well and then the optical density at 450 nm was measured using microplate reader.

2.5 | Colony formation assay

Eight hundred cells were seeded into 6-well plate and incubated for 14 days. Then, methanol was added to the plate to fix the colonies. Colonies were further stained with crystal violet and counted under microscope.

**FIGURE 1**  
*KCNQ1OT1* expression and its impact on overall survival of OC patients. (a) Expression of *KCNQ1OT1* in OC cells and normal cell line. (b) Effect of *KCNQ1OT1* on the overall survival of OC patients. *KCNQ1OT1*, Potassium Voltage-Gated Channel Subfamily Q Member 1 opposite strand/antisense transcript 1; OC, ovarian cancer

**FIGURE 2**  
*KCNQ1OT1* overexpression promoted cell proliferation, colony formation, and invasion. (a) *KCNQ1OT1* expression was detected following p*KCNQ1OT1* transfection. (b) CCK-8 assay to detect proliferation of cells transfected with p*KCNQ1OT1*. (c) Colony formation of cells after transferring of p*KCNQ1OT1*. (d) Cell invasion rate of cells with p*KCNQ1OT1* transfection was measured. CCK-8: cell counting kit-8; *KCNQ1OT1*, Potassium Voltage-Gated Channel Subfamily Q Member 1 opposite strand/antisense transcript 1; OC, ovarian cancer
2.6 | Transwell invasion assay

Cell invasion ability was analyzed with transwell invasion assay. $2 \times 10^5$ in serum-free medium was added into the upper chamber of 8 μm transwell unit (Corning, NY, USA) that pre-coated with Matrigel. RPMI-1640 supplemented with FBS was filled into the lower chamber. After incubation for 48 hr, invasive cells were fixed and stained. At length, the invasive cell numbers were counted under microscope.

2.7 | Luciferase reporter assay

IncBase was used to explore the potential miRNA target of KCNQ1OT1, and we identified MIR-142-5p was a potential target. TargetScan was utilized to investigate the potential target of MIR-142-5p, and we showed CAPN10 was a putative target. The wild-type sequence of KCNQ1OT1 and CAPN10 was inserted into pmirGLO to generate KCNQ1OT1-wt and CAPN10-wt. Site-direct mutagenesis kit was used to generate mutant luciferase vectors (KCNQ1OT1-wt and CAPN10-wt). Cells were co-transfected with luciferase vectors or miRNAs using Lipofectamine 2000. After 48 hr of transfection, relative luciferase activity was analyzed with Dual-luciferase reporter assay system (Promega, Madison, WI, USA).

2.8 | Prognostic values of KCNQ1OT1, MIR-142-5p, and CAPN10 on OC patients

The effects of KCNQ1OT1, MIR-142-5p, and CAPN10 on OC patients were explored at Kaplan-Meier plotter website (http://kmplot.com/analysis/index.php?p=background, Nagy, Lánczky, Menyhárt, & Győrffy, 2018).

2.9 | Statistical analysis

Results obtained from in vitro experiments were analyzed at Graphpad prism 7 (San Diego, CA, USA) and then presented as mean ± standard deviation (SD). Differences among groups were analyzed using Student's $t$-test or one-way Analysis of Variance and Tukey post-hoc test. $p < .05$ was believed as statistically significant.

3 | RESULTS

3.1 | Expression of KCNQ1OT1 was elevated in OC

qRT-PCR revealed that KCNQ1OT1 levels were significant higher in OC cell lines than in normal cell line (Figure 1a).
Survival analysis revealed that patients with high KCNQ1OT1 expression level tend to have worse prognosis compared to those with low KCNQ1OT1 expression level (Figure 1b).

3.2 | KCNQ1OT1 overexpression promotes OC cell proliferation and invasion in vitro

The SKOV3 cell line was selected for gain-of-function experiments. qRT-PCR revealed that KCNQ1OT1 levels could be significantly elevated by pKCNQ1OT1 (Figure 2a). CCK-8 assay and colony formation assay revealed that KCNQ1OT1 overexpression promoted OC cell growth (Figure 2b,c). Furthermore, transwell invasion assay showed the cell invasion of OC cell was promoted after KCNQ1OT1 overexpression (Figure 2d).

3.3 | Knockdown of KCNQ1OT1 inhibits OC cell proliferation and invasion in vitro

OVCAR3 cell line was used for loss-of-function experiments. si-KCNQ1OT1 introduction significantly decreased the levels of KCNQ1OT1 (Figure 3a). In vitro experiments revealed that knockdown the expression KCNQ1OT1 was able to inhibit OC cell proliferation, colony formation, and cell invasion (Figure 3b-d).

3.4 | Interaction of KCNQ1OT1 and MIR-142-5p in OC

IncBase showed that MIR-142-5p was a putative target for KCNQ1OT1 (Figure 4a). MIR-142-5p expression level was revealed to be decreased expression in OC cells compared with normal cell line (Figure 4b). Meanwhile, we showed that low MIR-142-5p was a predictor for poor overall survival of OC patients (Figure 4c). Luciferase activity reporter assay revealed that overexpression of MIR-142-5p inhibited the luciferase activity of cells transfected with KCNQ1OT1-wt (Figure 4d).

3.5 | Interaction of MIR-142-5p and CAPN10 in OC

Furthermore, TargetScan showed that CAPN10 was a putative target for MIR-142-5p (Figure 5a). qRT-PCR revealed

**FIGURE 4** KCNQ1OT1 functioned as a sponge for MIR-142-5p. (a) Binding model of KCNQ1OT1 and MIR-142-5p. (b) Expression of MIR-142-5p in OC cells and normal cell line. (c) Effect of MIR-142-5p on the overall survival of OC patients. (d) Luciferase activity in cells with luciferase vectors and miRNAs transfection. KCNQ1OT1, Potassium Voltage-Gated Channel Subfamily Q Member 1 opposite strand/antisense transcript 1; MIR-142-5p, MICRORNA-142-5p; miR-NC, negative control microRNA; mt: mutant; OC, ovarian cancer; wt: wild-type
that CAPN10 expression level in OC cells was higher than in normal cell line (Figure 5b). Kaplan–Meier plotter showed that high CAPN10 level indicates a poor prognosis of OC cancer patients (Figure 5c). Luciferase activity reporter assay indicated that overexpression of MIR-142-5p inhibits the relative luciferase activity of cells transfected with CAPN10-wt (Figure 5d).

3.6 | KCNQ1OT1 exerts its function through MIR-142-5p/CAPN10

qRT-PCR revealed that CAPN10 expression was significantly increased by pCAPN10 (Figure 6a). CCK-8 assay, colony formation, and transwell invasion assay showed that CAPN10 overexpression promotes OC cell growth and invasion (Figure 6b-d). In the meantime, we showed overexpression of MIR-142-5p inhibits OC cell proliferation, colony formation, and invasion (Figure 6b-d). Importantly, we showed KCNQ1OT1 targeting the MIR-142-5p/CAPN10 axis to regulate OC cell behaviors through rescue experiments (Figure 6b-d).

4 | DISCUSSION

LncRNA and miRNAs are RNA molecules that play crucial roles in multiples cellular processes including cell growth, metastasis, and drug resistant (Li & Zhan, 2019; Long, Song, et al., 2019; Qi et al., 2019). The competitive RNA (ceRNA) theory has connected lncRNA and miRNA together (Salmena, Poliseno, Tay, Kats, & Pandolfi, 2011). A recent work conducted by Zhao et al. revealed several lncRNA/miRNA/mRNA triplets that may contribute to the cisplatin-resistant in epithelial OC (Zhao, Tang, Zuo, Zhang, & Wang, 2019). Hence, the investigations on the aberrantly expressed molecules contributed to OC initiation and progression may help to better control OC in the future.

Previous studies have suggested that lncRNA KCNQ1OT1 plays crucial roles in cancer biology (Li et al., 2019; Xian & Zhao, 2019). In this work, we showed expression levels of lncRNA KCNQ1OT1 in OC cells were higher than in normal cell line. Through Kaplan–Meier plotter analysis, we showed high KCNQ1OT1 expression was correlated with poor overall survival of OC patients. Moreover, after KCNQ1OT1...
overexpression, we showed that cell proliferation, colony formation, and cell invasion of OC cells were significantly promoted. Besides that, after KCNQ1OT1 knockdown, cell proliferation, colony formation, and cell invasion of OC cells were significantly inhibited. Collectively, our work indicated that KCNQ1OT1 may have an oncogenic role in OC.

Bioinformatic analysis showed MIR-142-5p was a possible target for KCNQ1OT1. MIR-142-5p was reported to be a decreased expression in non-small cell lung cancer tissues and cells (Wang, Liu, Fang, & Yang, 2017). Overexpression of MIR-142-5p suppressed non-small cell lung cancer progression in vitro and in vivo via regulating phosphatidylinositol-4,5-bisphosphate 3-kinase, catalytic subunit alpha (171,834) (Wang et al., 2017). Moreover, MIR-142-5p was revealed to function as a bridge between KCNQ1OT1 and cyclin-dependent kinase 5 (123,831) to regulate lung adenocarcinoma cell migration, invasion, and epithelial mesenchymal transition (Jia et al., 2019). In this work, we showed MIR-142-5p expression was decreased in OC cells and correlated with poor overall survival of cancer patients. Luciferase activity reporter assay revealed the direct interaction of MIR-142-5p and KCNQ1OT1. Moreover, by exploring the target of MIR-142-5p, we identified a direct connection of MIR-142-5p and CAPN10. CAPN10 was found highly expressed in OC cells and predicted poor overall survival of cancer patients. CAPN10 belongs to the mitochondrial calpain system and reported to be regulated by oncogene GAEC1 (612,130) to promote tumor progression (Chan et al., 2013). One drawback of this work was the lack of in vivo experiments. Hence, we will perform animal experiments in the near future to validate the KCNQ1OT1/MIR-142-5p/CAPN10 axis in OC and to confirm their roles in regulating cancer progression.

To sum up, our results revealed that MIR-142-5p, sponged by KCNQ1OT1, target CAPN10 to affect the progression of OC. Our study provided novel insights on the understanding of OC progression and the potential to use KCNQ1OT1 as a target for OC therapy. However, further studies are needed to understand the clinical significance of KCNQ1OT1/MIR-142-5p/CAPN10 in OC.

CONFLICT OF INTEREST
The authors declare that they have no conflict of interest.

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