Long Non-Coding RNA Paternally Expressed Imprinted Gene 10 (PEG10) Elevates Diffuse Large B-Cell Lymphoma Progression by Regulating Kinesin Family Member 2A (KIF2A) via Targeting MiR-101-3p

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Background: Diffuse large B-cell lymphoma (DLBCL) is a common malignant tumor in the immune system with high mortality. We investigated the functional effects of long non-coding RNA paternally expressed imprinted gene 10 (PEG10) on DLBCL progression.

Material/Methods: Real-time quantitative polymerase chain reaction was used to measure the level of PEG10, kinesin family member 2A (KIF2A) and microRNA-101-3p (miR-101-3p) in DLBCL tissues and cell lines. The relative protein level was detected by western blot analysis. The biological behaviors including cell proliferation, apoptosis, migration, and invasion were determined by MTT assay, flow cytometry analysis, and Transwell assays, respectively. Bioinformatics analysis and dual-luciferase reporter assay were performed to evaluate the interaction among PEG10, miR-101-3p, and KIF2A.

Results: PEG10 and KIF2A level were significantly upregulated, while miR-101-3p was downregulated in DLBCL tissues and cells. PEG10 positively regulated KIF2A level in DLBCL. PEG10, or KIF2A deletion significantly inhibited the proliferative, migratory, and invasive abilities of DLBCL cells and elevated cell apoptosis in DLBCL cells. KIF2A upregulation partially reversed the effects of PEG10 downregulation on cell growth, metastasis, and apoptosis in DLBCL. Moreover, PEG10 negatively regulated miR-101-3p level and miR-101-3p upregulation exerted inhibition effects on the progression of DLBCL. Besides, miR-101-3p was a target of PEG10 and miR-101-3p could directly target KIF2A. PEG10 promoted KIF2A level by sponging miR-101-3p.

Conclusions: Our findings revealed that PEG10 played an oncogenic role in DLBCL progression, which might be a potential target for the treatment of DLBCL.

MeSH Keywords: Cell Proliferation • Lymphoma, B-Cell • MicroRNAs
Background

Diffuse large B-cell lymphoma (DLBCL) is a solid tumor of the immune system with a fast-growing incidence, accounting for 30% to 40% in non-Hodgkin lymphomas [1-3]. Previous studies reported that DLBCL frequently occurs in patients older than 60 and 70 years old [4]. The diagnosis for DLBCL patients is based on the clinical features, including a high degree of proliferation and strong metastasis, which resulted in highly variable treatment outcomes and prognosis for DLBCL patients [5]. The common lesion sites of the solid tumor DLBCL are mainly in the thymus, spleen, lymph nodes, and other lymphoid organs [6]. Genetic alternation, virus infection, and disorders of the immune system exerted crucial effects on the biological behaviors in the initiation and development of DLBCL. Although the diagnosis and treatment methods of DLBCL have achieved rapid development in recent years, there are still about 40% of DLBCL patients at an advanced stage fail due to remission and relapse, leading to the high mortality rate. The diagnosis biomarkers for early DLBCL patients remain lacking. Thus, it is of great importance to find efficient therapeutic targets for DLBCL patients.

Long non-coding RNAs (lncRNAs) with the length >200 nucleotides are a group of non-protein-coding RNAs that act as regulators in the processes of human cancers [7]. LncRNAs are involved in biological processes by interacting with DNA, RNA, and protein and by modulating the transcriptional or post-transcriptional expression level [8,9]. To date, accumulating evidence indicates that aberrantly expressed lncRNAs are closely related to the progression and prognosis of tumors [10]. Multiple research studies reported that dysregulation of lncRNAs was observed in DLBCL [11]. The LncRNA HULC deletion can attenuate cell growth in DLBCL cells by suppressing the level of cyclinD1 [12]. TUG1 has been identified as an oncogene in DLBCL, which could inhibit the degradation of MET and repress DLBCL cell growth and proliferation [13]. A previous study revealed that SNHG16 elevated the progression of DLBCL by boosting cell growth and inhibiting cell apoptosis through targeting miR-497-5p [14]. LncRNA paternally expressed imprinted gene 10 (PEG10) located on the chromosome 7q21 was first reported in 2001 [15]. PEG10 was confirmed to contribute to multiple functions including cell growth, differentiation, and apoptosis [16,17]. Additionally, PEG10 was involved in various malignancies, including DLBCL [18]. However, the molecular mechanism of PEG10 in DLBCL is still largely unknown.

PEG10 has been proven to function as competing endogenous RNA to sponge miRNAs and exert its functional effects. For example, PEG10 directly targeted miR-134 to regulate cell proliferation and metastasis in bladder cancer and affect the proliferative ability and apoptotic rate of HCT-116 cells via sponging miR-491 [19,20]. MicroRNA-101-3p (miR-101-3p) acted as a suppressor in bladder and gastric cancer [21,22]. However, the functional role of miR-101-3p in DLBCL is unclear. In the current research, starBase v2.0 predicted that kinesin family member 2A (KIF2A) contained the putative binding site of miR-101-3p. Thus, we determined PEG10, miR-101-3p, and KIF2A levels in DLBCL tissues, as well as in C1R-neo, SU-DHL-8, and OCI-LY-8 cells. Moreover, we aimed to explore the functional effects of PEG10 on DLBCL. Besides, the interaction among PEG10, miR-101-3p, and KIF2A in DLBCL was investigated. We attempted to find a potential therapeutic approach for DLBCL.

Material and Methods

Tissues samples

The 25 individuals with normal lymph nodes were used as controls and the 25 patients diagnosed with DLBCL participated in this study and signed informed consents. The normal individuals and the DLBCL patients from the Affiliated Shanxi Tumor Hospital of Shanxi Medical University had no prior local or systemic treatment. These experiments were approved by the Human Research Ethics Committee of Affiliated Shanxi Tumor Hospital of Shanxi Medical University.

Cell lines

DLBCL cell lines (SU-DHL-8 and OCI-LY-8) and normal lymphoblast cell line (C1R-neo) were used in this study. C1R-neo and SU-DHL-8 cells were obtained from American Type Culture Collection (Manassas, VA, USA) and OCI-LY-8 cells were bought from Shanghai Zishi Biological Technology Co., Ltd. (Shanghai, China). Streptomycin/penicillin (100 U/mL; Invitrogen, Carlsbad, CA, USA) and 10% fetal bovine serum (FBS; Invitrogen) were added into Roswell Park Memorial Institute 1640 (RPMI-1640) medium (Gibco, Carlsbad, CA, USA), which was used to culture cells at a humidified chamber at 37°C supplemented with 5% CO2.

Cell transfection

SiRNA against PEG10 (si-PEG10) or KIF2A (si-KIF2A) and the negative controls (si-NC), KIF2A overexpression vector (KIF2A) and the control (pcDNA), PEG10 overexpression vector (PEG10) and the blank control (pcDNA) were purchased from GenePharma (Shanghai, China). MiR-101-3p mimic (miR-101-3p) and control (miR-NC), miR-101-3p inhibitor (anti-miR-101-3p) and control inhibitor (anti-miR-NC) were bought from Ribobio (Guangzhou, China). All these vectors were transfected into DLBCL cells using Lipofectamine 2000 Reagent (Invitrogen).

Real-time quantitative polymerase chain reaction (RT-qPCR)

The isolation of total RNA from DLBCL tumor tissues and cells was carried out using TRIzol reagent (Invitrogen). The level
of PEG10, miR-101-3p, and KIF2A was analyzed by RT-qPCR analysis using an ABI 7500 Real-time PCR system (Bio-Rad Laboratories Inc., Hercules, CA, USA). The levels of miR-101-3p, PEG10, and KIF2A were normalized by U6 and glyceraldehyde 3-phosphate dehydrogenase (GAPDH), respectively. The specific primers were as follows:

PEG10: CACCTTCCTCCTGGTCCGC (sense) and CCTCTGCCACTCCCTCTTTT (antisense),
GAPDH: TGGTATGTCGAAAGGACTCA (sense) and CACATAGGCGAGGATGAT (antisense),
mir-101-3p: GCCGCGCATACAGTACTGTGATA (sense) and GCCGCCAGTTGTCAGACTAC (antisense),
KIF2A: GCCCTTGATGACTCAGCTCC (sense) and TCTCTGAAAGTGCAACCCC (antisense),
U6: TCCGATCTGGAACGGTTC (sense) and GTGACGGTGTGTAGGTT (antisense).

The 2^-ΔΔCT method was used to calculate the level of PEG10, miR-101-3p, and KIF2A.

Western blot analysis

Total protein was extracted using RIPA lysis buffer (Beyotime, Shanghai, China). The isolated total protein was separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE). The primary antibodies including anti-KIF2A (1: 500, ab197998, Abcam, Cambridge, UK), anti-Bax (1: 1000, ab32503, Abcam), anti-caspase-3 (1: 500, ab4051, Abcam), anti-Bcl-2 (1: 1000, ab59348, Abcam), anti-cyclinD1 (1: 200, ab16663, Abcam), and GAPDH (1: 1000, ab8245, Abcam) were used in this study. Secondary antibodies included goat-anti-rabbit IgG H&L (1: 2000, ab6721, Abcam) and goat anti-mouse IgG H&L (1: 2000, ab205719, Abcam). Finally, the protein signals were measured by the enhanced chemiluminescence kit.

Cell proliferation assay

DLBCL cells were transfected for 48 hours. Then, cells were seeded in 96-well plates and cultured at the humidified chamber for 24, 48, and 72 hours. Then 20 μL MTT reagent (5 mg/mL) was added into the medium for 4 hours. Subsequently, the blue crystal in cells was dissolved by dimethyl sulfoxide (150 μL per well). Finally, cell viability was measured using a microplate reader at 490 nm.

Cell apoptosis assay

After transfection, the binding buffer was used to resuspend the DLBCL cells (1 x 10^6 cells/mL). Then, SU-DHL-8 and OCI-LY-8 cells were double stained with 5 μL Annexin V-fluorescein isothiocyanate (FITC) and propidium iodide (PI) (BestBio, Shanghai, China). Cell apoptosis was measured by a FACSscan flow cytometer (Becton Dickinson, Mountain View, CA, USA).

Transwell assay

For examination of the migrated and invasive cells, SU-DHL-8 and OCI-LY-8 cells mixed with the serum-free medium were seeded on the upper chamber (Corning Incorporated, Big Flats, NY, USA) pre-coated with Matrigel for the invasion assay, while the chambers were non-coated with Matrigel in the Transwell migration assay. DMEM with 10% FBS was provided for the lower chamber, regarding as a chemoattractant. The not-migrated or not-invaded cells on the top surface were wiped off using a cotton swab, whereas the migrated or invaded cells on the lower chamber were fixed, stained, and then counted using a microscope.

Dual-luciferase reporter assay

The 3'UTR fragment of KIF2A (KIF2A-3'UTR-WT) and the wide-type PEG10 sequence (WT-PEG10) containing the predictive binding sites of miR-101-3p were inserted into pGGL3 promoter vectors (Invitrogen). The mutant sequences of PEG10 and KIF2A were used to establish PEGL0-mutant (MUT-PEG10) and KIF2A-mutant (KIF2A-3'UTR-MUT) reporter vectors. Then, SU-DHL-8 and OCI-LY-8 cells were co-transfected with miR-NC or miR-101-3p mimics and MUT-PEG10, WT-PEG10, KIF2A-3'UTR-WT or KIF2A-3'UTR-MUT. Finally, the dual-luciferase reporter assay system (Promega, Madison, WI, USA) was carried out to determine the luciferase activity in SU-DHL-8 and OCI-LY-8 cells.

Statistical analysis

The data in the present research were displayed as the mean±the standard deviations (SD). Student's t-test for comparison between 2 groups and one-way analysis of variance (ANOVA) for differences among more than 2 groups followed by Turkey’s test were used. The interaction between variables was analyzed by Pearson correlation analysis. The value of P<0.05 means a statistically significant difference.

Results

PEG10 and KIF2A were upregulated in DLBCL tissues and cells

RT-qPCR indicated that the level of PEG10 and KIF2A was boosted in DLBCL tissues (Figure 1A, 1B). As shown in Figure 1C, KIF2A was highly expressed in DLBCL tissues relative to that in normal tissues. Moreover, we also performed RT-qPCR and western blot assay to determine the level of PEG10 and KIF2A in C1R-neo, SU-DHL-8, and OCI-LY-8 cells. PEG10 and KIF2A levels were significantly increased in SU-DHL-8 and OCI-LY-8 cells compared with C1R-neo cells (Figure 1D). Similarly, the protein level of KIF2A was markedly enhanced in DLBCL cells.
Besides, we found that there was a positive relationship between PEG10 level and KIF2A level (Figure 1F). Taken together, these data indicated that PEG10 and KIF2A played vital roles in DLBCL.

**PEG10 deletion inhibited cell growth and metastasis and enhanced cell apoptosis in DLBCL**

We explored the functional effects of PEG10 on DLBCL. SU-DHL-8, and OCI-LY-8 cells were transfected with si-NC or si-PEG10. The data showed that PEG10 deletion significantly suppressed the level of PEG10 in both SU-DHL-8 and OCI-LY-8 cells (Figure 2A). Knockdown of PEG10 markedly inhibited cell proliferation in both SU-DHL-8 and OCI-LY-8 cells (Figure 2B). Flow cytometry analysis indicated that cell apoptosis was significantly enhanced by downregulating PEG10 in DLBCL cells (Figure 2C). As described in Figure 2D, cyclinD1 and Bcl-2 were decreased, while the level of caspase-3 and Bax was significantly increased in both SU-DHL-8 and OCI-LY-8 cells transfected with si-PEG10. The results suggested that knockdown of PEG10 could significantly suppress cell metastasis in DLBCL cells (Figure 2E). All these results demonstrated that PEG10 promoted the progression of DLBCL.

**KIF2A deletion suppressed cell proliferation, migration, invasion and elevated cell apoptosis in DLBCL**

Firstly, we measured the knockdown efficiency of si-KIF2A in DLBCL. The data indicated that the mRNA level of KIF2A was markedly alleviated in SU-DHL-8 and OCI-LY-8 cells after transfection of si-KIF2A (Figure 3A). Similarly, we discovered that knockdown of KIF2A could repress the protein level of KIF2A (Figure 3B, C). We further demonstrated that knockdown of KIF2A significantly suppressed cell proliferation, migration, and invasion in DLBCL (Figure 3D, E). Flow cytometry analysis revealed that cell apoptosis was increased in both SU-DHL-8 and OCI-LY-8 cells transfected with si-KIF2A (Figure 3F). All these results indicated that knockdown of PEG10 could repress the protein level of KIF2A.
MTT assay indicated that KIF2A deletion significantly inhibited cell proliferation in both SU-DHL-8 and OCI-LY-8 cells (Figure 3C). Cell apoptosis was significantly promoted by downregulating KIF2A in DLBCL cells (Figure 3D). Moreover, the proliferation and apoptosis related proteins were measured by western blot analysis. The apoptosis-related protein Bcl-2 and the proliferation-related protein cyclinD1 were repressed by KIF2A knockdown, whereas caspase-3 and Bax were highly expressed in DLBCL cells transfected with si-KIF2A (Figure 3E). Besides, we observed that the migratory and invasive abilities were significantly attenuated in SU-DHL-8 and OCI-LY-8 cells transfected with si-KIF2A (Figure 3F, 3G). All these data suggested that KIF2A deletion suppressed the progression of DLBCL. 

KIF2A overexpression reversed the effects of PEG10 knockdown on DLBCL progression

Using RT-qPCR, we confirmed that PEG10 deletion decreased the level of KIF2A, while KIF2A was highly expressed in both SU-DHL-8 and OCI-LY-8 cells transfected with KIF2A overexpression.
vector (Figure 4A). Similarly, the protein level of KIF2A was suppressed by PEG10 downregulation and enhanced by KIF2A overexpression in DLBCL cells (Figure 4B). KIF2A overexpression reversed the inhibitory effect of PEG10 deletion on cell proliferation in both SU-DHL-8 and OCI-LY-8 cells (Figure 4C). Our data also noted that PEG10 deletion induced cell apoptosis was markedly attenuated by overexpression of KIF2A (Figure 4D).

In Transwell assay, we discovered that the inhibitory effects of PEG10 deletion on cell migration and invasion were reversed by upregulating KIF2A (Figure 4E, 4F). Taken together, upregulation of KIF2A could reverse the effects of PEG10 knockdown on cell growth, apoptosis and metastasis in DLBCL.

MiR-101-3p overexpression inhibited cell growth, metastasis and enhanced cell apoptosis in DLBCL

As shown in Figure 5A and 5B, miR-101-3p level was significantly repressed in DLBCL tissues and cells compared with the level in normal tissues and cells. Moreover, we discovered a negative relationship between PEG10 level and miR-101-3p level (Figure 5C). The level of miR-101-3p was boosted in DLBCL cells transfected with miR-101-3p mimics (Figure 5D). MiR-101-3p overexpression significantly inhibited the proliferative ability of SU-DHL-8 and OCI-LY-8 cells (Figure 5E). However, the apoptosis rate of DLBCL cells was markedly elevated by upregulating miR-101-3p (Figure 5F). Increased level of miR-101-3p suppressed cell migration and invasion in both SU-DHL-8 and OCI-LY-8 cells (Figure 5G). We also found that miR-101-3p upregulation decreased the level of cyclinD1 and Bcl-2 and boosted the level of Bax and caspase-3 in DLBCL cells (Figure 5H). Overall, miR-101-3p might play a suppressor role in DLBCL.

PEG10 regulated KIF2A level by sponging miR-101-3p

MiRcode predicted that there were potential binding sites between PEG10 and miR-101-3p, while starBase v2.0 showed that KIF2A might be a target of miR-101-3p (Figure 6A). The results noted that the luciferase activity was significantly decreased

Figure 3. KIF2A deletion suppresses cell growth, metastasis and elevates cell apoptosis in DLBCL. (A) KIF2A level was detected by RT-qPCR. (B) The protein level of KIF2A was measured by western blot. (C) MTT assay for cell proliferation. (D) Flow cytometry for cell apoptosis. (E) Western blot analysis for the determination of cyclinD1, Bcl-2, caspase-3 and Bax level. (F, G) Transwell assays for cell migration and invasion. * P<0.05. KIF2A – kinesin family member 2A; DLBCL – diffuse large B-cell lymphoma; RT-qPCR – real-time quantitative polymerase chain reaction.
in DLBCL cells co-transfected with miR-101-3p mimic and WT-PEG10 (Figure 6B). Moreover, miR-101-3p was inversely regulated by PEG10 in both SU-DHL-8 and OCI-LY-8 cells (Figure 6C). We also discovered that miR-101-3p mimic repressed the luciferase activity in DLBCL cells transfected with KIF2A-3’UTR-WT. However, for cells transfected with KIF2A-3’UTR-MUT, the luciferase activity was not changed in the miR-101-3p group in comparison to the control group (Figure 6D). MiR-101-3p overexpression inhibited KIF2A protein level, while PEG10 upregulation reversed the inhibitory effect of miR-101-3p overexpression on KIF2A level in both SU-DHL-8 and OCI-LY-8 cells (Figure 6E). All these data demonstrated that PEG10 regulated the protein level of KIF2A by targeting miR-101-3p in DLBCL.

**PEG10 regulated cell proliferation and apoptosis via miR-101-3p/KIF2A axis in DLBCL**

PEG10 knockdown significantly decreased cyclinD1 and Bcl-2 level and facilitated caspase-3 and Bax level in DLBCL cells, which were eliminated after the transfection of miR-101-3p inhibitor or overexpression of KIF2A (Figure 7A, 7B). These data suggested that PEG10 regulated KIF2A to further affect the levels of proliferation and apoptosis related proteins, which played crucial roles in DLBCL cell growth and apoptosis.

**Discussion**

Previous researches demonstrated that IncRNAs participated in DLBCL development [12,23]. Moreover, PEG10 was demonstrated to play a vital role in various cancers. For instance, PEG10 level was markedly upregulated in bladder cancer and pancreatic cancer and enhanced the abilities of cell growth, as well as cell metastasis in bladder cancer cells and pancreatic cancer cells *in vitro*, which might be a prognosis biomarker [24,25]. The similar phenomenon was discovered in breast cancer, the proliferative ability of breast cancer cells was significantly enhanced by PEG10 [26]. Zang et al. indicated that PEG10 made a contribution to cell invasion and proliferation in esophageal cancer [27]. However, the level of PEG10 in DLBCL and its functional role remain largely uninvestigated. In this research, we discovered the high level of PEG10 in DLBCL.
tumor tissues. Similarly, upregulated level of PEG10 was discovered in DLBCL cells in comparison to normal B lymphocytes. Moreover, PEG10 deletion suppressed cell proliferation and promoted the apoptotic rate in DLBCL cells. Overall, knockdown of PEG10 exerted a suppressive role in the biological processes of DLBCL. Subsequently, we explored the underlying mechanism of PEG10 in the progression of DLBCL.

MicroRNAs (miRNAs) could regulate the translation of mRNAs through binding to mRNAs 3'-UTR [28]. Accumulating evidence
demonstrated that miRNAs dysregulation were closely correlated with various cancers initiation and development [29]. It has been reported that the prognosis of DLBCL patients might be associated with the specific miRNAs [30]. For example, miR-155 might be a potential prognosis biomarker in DLBCL [31]. He et al. reported that miR-195 upregulation could alleviate the immune escape of DLBCL and regulate the release of interferon gamma (IFN-γ), interleukin-10 (IL-10), tumor necrosis factor alpha (TNF-α) in PD-1T cells [32]. Besides, the aberrant levels of miR-22, miR-23a, and miR-4638-5p were also observed in DLBCL [33–35]. We observed that miR-101-3p was upregulated in DLBCL tumor tissues and cells. Moreover, we discovered a negative relationship between miR-101-3p level and PEG10 level. MiR-101-3p acted the inhibitory effects on cell proliferation and metastasis and increased the apoptotic rate of SU-DHL-8 and OCI-LY-8 cells. Besides, miR-101-3p was testified as a target of PEG10 in DLBCL.

**Figure 6.** PEG10 regulates KIF2A level by sponging miR-101-3p. (A) MiRcode showed the putative binding sites between PEG10 and miR-101-3p, while starBase2.0 predicted that the KIF2A mRNA 3’UTR sequence contained the binding sites of miR-101-3p. (B) The luciferase activity in DLBCL cells co-transfected with miR-NC, miR-101-3p, WT-PEG10, and MUT-PEG10 was detected by dual-luciferase reporter assay. (C) RT-qPCR was used to examine miR-101-3p level in DLBCL cells transfected with pcDNA, PEG10, si-NC, and si-PEG10. (D) Detection of the luciferase activity in DLBCL cells co-transfected with miR-NC, miR-101-3p, KIF2A-3’UTR-WT, and KIF2A-3’UTR-MUT by dual-luciferase reporter assay. (E) The protein KIF2A level was detected by western blot analysis in DLBCL cells transfected with miR-NC, miR-101-3p, miR-101-3p+pcDNA or miR-101-3p+PEG10.

* P<0.05. PEG10 – paternally expressed imprinted gene 10; KIF2A – kinesin family member 2A; DLBCL – diffuse large B-cell lymphoma; RT-qPCR – real-time quantitative polymerase chain reaction.
KIF2A is a member of kinesin superfamily proteins with the onco-
genic function involving in human cancers [36]. The regulatory role
of KIF2A in carcinogenesis of various tumors has received exten-
sive attention. Zhao et al. reported that KIF2A deletion exerted cell
growth and invasion repression effects in MKN-45 and SGC-7901
cells through repressing the level of MTI-MMP [37]. Furthermore,
KIF2A was increased in lung adenocarcinoma and the high level
of KIF2A played a promotion role in cell growth and metastasis
of lung adenocarcinoma cells, which might serve as an effective
prognosis biomarker for lung adenocarcinoma [38]. Zhang et al.
noted that enhanced level KIF2A was discovered in DLBCL that
related to the prognosis of DLBCL [39]. Our data suggested that
KIF2A was upregulated in DLBCL. The effects of KIF2A knockdown
hibited cell growth and metastasis and induced cell apoptosis in
SU-DHL-8 and OCI-LY-8 cells. Besides, we observed that PEG10
could regulate KIF2A level by sponging miR-101-3p in DLBCL cells.
PEG10 deletion could suppress cell growth, metastasis, and pro-
moted cell apoptosis through miR-101-3p/KIF2A axis in DLBCL.

Conclusions

In conclusion, we discovered that PEG10 and KIF2A level were
markedly increased, while miR-101-3p was decreased in DLBCL
tumor tissues and cells. Functionally, PEG10 deletion signifi-
cantly suppressed cell proliferation and metastasis, and en-
hanced cell apoptosis in DLBCL cells. Moreover, our findings
demonstrated that PEG10 directly targeted miR-101-3p and
that miR-101-3p was a target of KIF2A. Besides, we found that
PEG10 regulated cell proliferation, apoptosis, and metastasis
through the miR-101-3p/KIF2A axis, providing a new therapeu-
tic target for the diagnosis and treatment of DLBCL.

Conflicts of interest.

None.


