Revista Argentina de Clínica Psicológica
2020, Vol. XXIX, N°4, 80-86
DOI: 10.24205/03276716.2020.808



# Effects of lncRNA-LET targeting miR-93-5p on proliferation and apoptosis of human dermal fibroblasts after ultraviolet radiation

**Wenqiang Zhang[a], Qian Ding[b*], Na Zhang[c]**

**Abstract**

**Objective:** To explore the effects of circular ribonucleic acid G-protein subunit beta 1 (circGNB1) on the migration, invasion and apoptosis of colon cancer cells and its molecular mechanism.

**Objective:** To evaluate the effects of long non-coding RNA (lncRNA)-low expression in tumor (lncRNA-LET) on the proliferation and apoptosis of human dermal fibroblasts (HDFs) after ultraviolet (UV) radiation and relevant molecular mechanism.

**Methods:** Primary HDFs were radiated with UV ray (5 J·cm-2), and the expression levels of lncRNA-LET and micro RNA (miR)-93-5p were measured through fluorescence real-time quantitative polymerase chain reaction (RT-qPCR). HDFs were separately transfected with small interfering (si)-LET, si-negative control (NC), miR-93-5p, miR-NC, anti-miR-NC + si-LET, and anti-miR-93-5p + si-LET and after UV radiation. The cell proliferation viability, cell apoptosis rate, and expressions of Cyclin D1 and cleaved-caspase-3 were detected by cell counting kit-8 assay, flow cytometry and Western blotting, respectively. The dual luciferase reporter assay and RT-qPCR were conducted to verify the targeted regulatory relationship of lncRNA-LET with miR-93-5p.

**Results:** The expression level of lncRNA-LET in HDFs after UV radiation rose, while that of miR-93-5p declined ($P<0.05$). After interference of lncRNA-LET expression or miR-93-5p overexpression, the HDFs after UV radiation exhibited increased proliferation rate and Cyclin D1 expression, and decreased apoptosis rate and cleaved-caspase-3 expression ($P<0.05$). On the contrary, the HDFs after UV radiation displayed lowered proliferation rate and Cyclin D1 expression, and raised apoptosis rate and cleaved-caspase-3 expression after suppressing both miR-93-5p and lncRNA-LET ($P<0.05$). The lncRNA-LET targeted miR-93-5p and negatively regulated the expression of miR-93-5p.

**Conclusion:** Interference of lncRNA-LET can facilitate the proliferation and repress the apoptosis of HDFs after UV radiation by targeting and regulating miR-93-5p expression.

**Key words:** lncRNA-LET, miR-93-5p, UV, dermal fibroblast, cell proliferation, apoptosis.

## 1. Introduction

Ultraviolet (UV) functions importantly in skin photoaging and dermal carcinogenesis. Studies have manifested that long-wave UV (UVA) can penetrate the dermis of the skin, inducing DNA damage, oxidative stress and programmed cell death of skin cells, and thereby destroying the structure of skin tissues and resulting in various skin diseases and even skin cancers (Liu X. et al., 2018; Greinert R. et al., 2012). The non-encoding RNAs are crucial epigenetic control molecules, particularly long non-encodable RNAs (lncRNAs

*a.Department of Clinical Teaching and Research Section, School of Nursing and Health College of Zhengzhou University, Zhengzhou, Henan, China;*
*b.Department of Thoracic Surgery, Henan Province People's Hospital, Zhengzhou, Henan, China;*
*c.Department of Neurosurgery, The First Affiliated Hospital of Zhengzhou University, Zhengzhou, Henan, China*
*Corresponding author: Qian Ding*
*Address: Department of Thoracic Surgery, Henan Province People's Hospital, Zhengzhou, Henan, China;*
*Email: daoyong13060@163.com*

with over 200nt lengths) and microRNAs (miRNAs with over 20nt lengths). Study has shown that UV induced human dermal fibroblast (HDF) damage can be linked to lncRNA's and miRNA's dysregulation (Zcheng Y. et al., 2017; Zheng Y. et al., 2017). In the 15q24.1 region, the low tumor expression of the lncRNA-LET may be inhibited in cancer cells in the case of malignant tumors such as the esophagus squamous carcinoma and lung adenocarcinoma (Wang P.L. et al., 2016; Liu B. et al., 2016). It has been established that there is opportunity for bioinformatics research

The lncRNA-LET linking area is miR-93-5p. MiR-93-5p has been shown to be active in inflammatory skin damage due to a medium-wave UV ray (UVB) and is a possible therapeutic option for skin injuries triggered by UVBs (Zhang N. et al., 2019). However, it remains unclear if miR-93-5p could be attacked by lncRNA-LET as a defensive factor in UV injury. And the expressions of lncRNA-LET in HDF and miR-93-5p in UV radiation have been further studied in this analysis in order to establish novel strategies to treat UV-induced HDF harm in lncRNA-LET aimed at miR-93-5p in HDF proliferation and apoptosis after UV radiation.

## 2. Materials and methods

### 2.1 Experimental materials

The Gibco (USA) has been acquired for Fetal Bovine Serum (FBS) and Dulbecco's modified Eagle Medio (DMEM). The BGI (Beijing, China) has been strengthened and distributed with Release, tiny interfere with RNA (Si-LET), scrambling negctive nonsense control (Si-NC), mime negative control (MiR-NC), Mir-93-5p inhibitor (Anti-MiR-NC) and Wild-type / Free reporter gene vector (TW / MUT-lncRNA-LET). The reverse transcriptase of M-MLV and the Master Mix (qPCR) of GoTaq® is obtained from Promega (USA). Nanjing Keyuan Biological Co., Ltd. obtained the Annexin V-fluorescein isothiocyanate / propidium iodide (Annexin V-FITC / PI) detection package. The Shanghai Beyotime Biotechnology Co., Ltd. purchased a Kit-8 (CCK-8) Cell Counting, a Radiosafety Assay (RIPA) buffer, Pro-assay Package for Bicinchoninic Acido (BCA), Cyclin D1 antibody for mouse, Caspasa Radbit-3 antibody rabbits, β-actin mouse antikota, Goat Immunoglobulin G(IgG) and Goat Anti-RabbitIgG.

### 2.2 Cell culture and grouping

The HDF was extracted from the dermis of the prepuce of 6-8 years old girls. Then the cells were harvested and developed in a humidified incubator with DMEM 10 percent and 5 percent CO2 at 37 ° C at second to fourth passage. The cultivation of the HDFs was typically used as the search party, while the 5 J·cm-2 UVA-cultivated HDFs were used as the UVA. UVA HDFs transfected with si-LET (group of SI-LETs), SI-NC (community SI-NC), miR-93-5p (community of MIR 93-5p), MIRO-NC (group of MIR-NC) and Ant-MIR- 93-5p + SI-LET (group of MIR-NC+ SI-LETs, group of MIR-NC+ Ant-MIR-NC+SI-LET) respectively followed by UV radiation.

### 2.3 Measurement of lncRNA-LET and miR-93-5p expressions by real-time quantitative PCR (RT-qPCR)

Absolute RNAs in each community utilizing the TRIzol reagent have been derived from the HDFs. The next move is to transcrib M-MLV reverted transcriptase to cDNAs for the complete protein. RT-qPCR was subsequently conducted on an ABi Prism Framework 7500 utilizing the GoTaq® qPCR Master Blend. Finally, lncRNA-LET expression levels (with internal comparison β-actin) and miR-93-5p (internal referencing U6) were examined using the 2-to-CT process. The first series is the following. Upstream main for LnkRNA LET: 5′-AGCGTTTACTTGTGTGT-3′′, downstream first: 5′-CAGAATGAATGATAGGAGC-3′′; upstream main for β-actin: 5′-CACTGTGCCTACGAGG-3′′, first downstream: 5′—TAATGCACGATTTTCc-3′′; first upstream for U6: 5′-GCTTGCCAGCACTATACT-3′′, downstream: 5′-GTGCAGGTCGTCGATTTC′′′′; first for

### 2.3 Detection of cell viability by CCK-8 assay

A total of 5×103 HDFs were inoculated into a 96- Right, well. Very nice. When cell confluence was 70 percent, various therapies were obtained independently in multiple study categories. In each cell, 10 μL CCK-8 was then introduced, accompanied by a 40 min reaction in the incubator. The cell survival rate was then determined as follows, using a microplate reader at 450 nm, with (A) absorption rate of each well: cell proliferation rate = (Aexperimental community – Ablank group)/ (Supervisory community -Ablank groups) = 100%.

### 2.4 Detection of cell apoptosis by flow cytometry

A limit of five to ten4 HDFs have been gathered and centrifuged and a surnatant has been extracted. Then, 1 mL of pre-cooled PBS was applied to suspend the cells and gently shook. The supernatant was dismissed after centrifugation again. If the steps above have been replicated in 200 μL of binding buffer, the cells have been loaded with 10 μL of Annexin V-FITC and well-mixed and the reaction has been 4 ° C for 30

minutes. Then the binding buffer was applied up to 300 µL, accompanied by a measurement of cell apoptosis within 1 h, and the pi 5 µL is equally matched.

**2.5 Measurement of Cyclin D1 and cleaved-caspase-3 protein expressions by Western blot**

The cells were isolated using RIPA buffer to collect their whole protein content, and the BCA protein test collection was used to assess its content. The proteins were then isolated and moved to a polyvinyllidene fluoride membrane by sodium sulfate-polyacrylamide gel electrophoresis. The membrane was then blocked at 60 minutes with a 5% bovine serum albumin, thus incubated at 4 ° C over midday with the main anticorpus solution (Cyclin D1: 1:200 diluted, and 1:1000 diluted with cleaved caspase-3 and β-actin). First, a color production chemical chromogenic reagent has been developed. Since taking photos in a gel imaging system, the gray value of each protein band was analyzed, and the relative expression level of target protein was calculated as follows: the relative expression level of target protein = gray valuetarget protein / gray valueβ-actin.

**2.6 Dual luciferase reporter assay**

The WT and MUT lncRNA-LET reporter gene vectors were developed in turn, called WT-lncRNA-LET, and MUT-lncRNA-LET, for the aiming impact of LNCRNA-LET on MiR-93-5p. Then the HDFs were co-transfected using the technique of liposome transfection by miR-93-5p mimics and WT-lncRNA-LET or MUT-lncRNA-LET. Cells were extracted at 48 hours after transfection and improvements in their luciferase behavior were observed in a twofold method of luciferase reporter experiments.

**2.7 Statistical analysis**

Triplicate and three times all the tests were conducted. The statistical study of all data was performed by SPSS 19.0, and the average variance was ± norm ('x±s). The comparisons were made between two groups and between many groups were carried out using a one-way variance study. An additional SNK-q research was carried out for Intergroup comparisons. A statistically important P<0.05 was found.

**3. Results**

**3.1 LncRNA-LET and miR-93-5p expressions in HDFs after UV radiation**

The expression level of lncRNA-LET in HDFs after UV radiation rose, while that of miR-93-5p declined (P<0.05) (Table 1).

**2.5 Effects of interfering lncRNA-LET expression on proliferation and apoptosis of HDFs after UV radiation**

The expression level of lncRNA-LET in HDFs after UV radiation was significantly lower in si-LET group as in the si-NC (P<0.05 group) the si-LET has been successfully transferred and the lncRNA-LET expression in HDFs has been suppressed after UV radiation. In comparison to si-NC, the si-LET groups displayed substantially higher rates of proliferation and cycline D1 levels of protein expression, and slightly lowered rate of apoptosis and cleaved-caspase-3 protein expression after UV (P<0.05) in HDFs (Table 2 and Figure 1)

*Table 1.* **LncRNA-LET and miR-93-5p expressions in HDFs after UV radiation ( $\bar{x}$ ± s, n=9)**

| Group | LncRNA LET | miR-93-5p |
|---|---|---|
| Control | 1.00±0.10 | 1.02±0.12 |
| UVA | 3.86±0.35* | 0.45±0.04* |
| t | 23.571 | 13.519 |
| P | 0.000 | 0.000 |

Compared with control, *P<0.05.

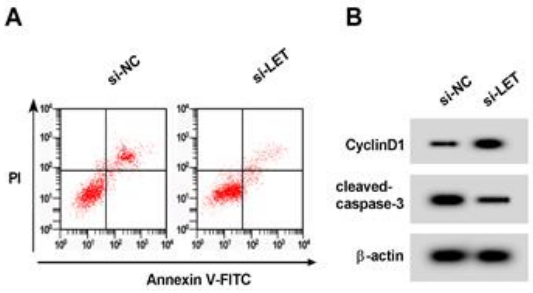


Figure 1. **Effects of interfering lncRNA-LET expression on proliferation and apoptosis of HDFs after UV radiation.**

A: Cell apoptosis detected by flow cytometry; B: Cyclin D1 and cleaved-caspase-3 protein expressions measured by Western blot.

*Table 2.* **Effects of interfering lncRNA-LET expression on proliferation and apoptosis of HDFs after UV radiation ( $\bar{x}$ ± s, n=9)**

| Group | LncRNA LET | CyclinD1 | Cleaved-caspase-3 | Proliferation rate (%) | Apoptosis rate (%) |
|---|---|---|---|---|---|
| si-NC | 1.00±0.10 | 0.35±0.03 | 0.96±0.09 | 100.28±10.04 | 30.25±3.02 |
| si-LET | 0.38±0.04* | 0.82±0.08* | 0.42±0.04* | 152.17±1.52* | 10.01±1.05* |
| t | 17.270 | 16.503 | 16.449 | 15.330 | 18.991 |
| P | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |

Compared with si-NC, *P<0.05.

**2.1. Effects of miR-93-5p overexpression on proliferation and apoptosis of HDFs after UV radiation**

The degree of expression of miR-93-5p in HDFs following UV radiation in miR-93-5p community was substantially higher than in miR-NC, exceeding the regulated miR-93-5p expressions in HDFs. rayons UV. UV. Compared to the miR- NC community, miR-93-5p showed slightly higher levels of Cyclin-D1 proliferation and protein expression in HDFs after UV radiation (P<0,05), substantially decreased apoptosis and cleaved-caspase-3 protein expression (Table 3 and Figure 2).

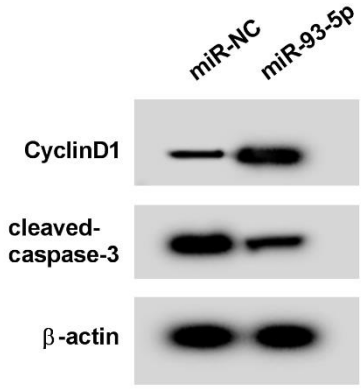


Figure 2. **Cyclin D1 and cleaved-caspase-3 protein expressions measured by Western blot.**

*Table 3.* **Effects of miR-93-5p overexpression on proliferation and apoptosis of HDFs after UV radiation ( $\bar{x}$ ± s, n=9)**

| Group | miR-93-5p | CyclinD1 | Cleaved-caspase-3 | Proliferation rate (%) | Apoptosis rate (%) |
|---|---|---|---|---|---|
| miR-NC | 1.02±0.10 | 0.32±0.03 | 0.95±0.08 | 100.86±10.01 | 29.02±2.85 |
| miR-93-5p | 2.69±0.25* | 0.78±0.08* | 0.37±0.04* | 142.05±14.20* | 9.21±0.90* |
| t | 18.607 | 16.152 | 19.454 | 7.113 | 19.885 |
| P | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |

Compared with miR-NC, *P<0.05.

**2.2. LncRNA-LET targetedly regulated miR-93-5p expression**

2.3. Based on the data from the online prediction of target genes using starBase tools, the lncRNA-LET nucleotide sequences and miR-93-5p are binding complementary (Figure 3). The findings of a dual luciferasis study showed that the luciferase activity of HDFs with miR-93-5p and WT-lncRNA-LET trans-transfected by miR-NC and WT-lncRNA-LET (P<0.05) was substantially reduced compared with that of HDFs with mir-93-5p and MUT-lncRNA-LET with luciferasis showed no major changes compared with that MIR-93-5p expression level was slightly lower in HDFs in si-LET than in miR-NC units, compared with the amount in si-LET in si-NCs (P<0.05) (Table 5).

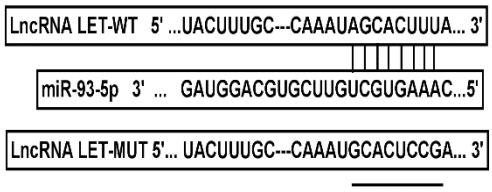


Figure 3. **Binding between nucleotide sequences of lncRNA-LET and miR-93-5p predicted by starBase software.**

*Table 4.* **Dual luciferase reporter assay results ( $\bar{x}$ ± s, n=9)**

| Group | Luciferase activity | |
|---|---|---|
| | WT-LncRNA LET | MUT-LncRNA LET |
| miR-NC | 1.00±0.10 | 1.02±0.11 |
| miR-93-5p | 0.48±0.04* | 1.01±0.12 |
| t | 14.484 | 0.184 |
| P | 0.000 | 0.856 |

Compared with miR-NC, *P<0.05.

*Table 5.* **Expression of miR-93-5p measured by RT-qPCR ( $\bar{x}$ ± s, n=9)**

| Group | miR-93-5p |
|---|---|
| pcDNA-NC | 1.02±0.11 |
| pcDNA-LET | 0.45±0.04* |
| si-NC | 1.01±0.10 |
| si-LET | 1.42±0.14# |
| F | 132.166 |
| P | 0.000 |

Compared with pcDNA-NC, *P<0.05; compared with si-NC, #P<0.05.

**2.4. Inhibiting miR-93-5p partly reversed the effects of lncRNA LET expression on proliferation and apoptosis of HDFs after UV radiation**

Compared with the miR-93-5p expresses in the anti-miR-NC + si-LET group, cell propagation and cyclin D1 protein level were significantly reduced in HDF after UV radiation, and the cell apoptosis and cloaved-caspase-3 levels of protein expression in the non-micronutrient R-93-5p + si-LET (P<0.05) groups were significantly increased (Table 6 and Figure 4).

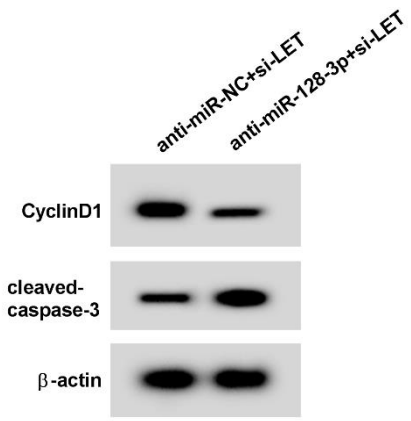


Figure 4. **Cyclin D1 and cleaved-caspase-3 protein expressions measured by Western blot.**

*Table 6.* **Low miR-93-5p expression partly reversed the effects of lncRNA LET expression on proliferation and apoptosis of HDFs after UV radiation ( $\bar{x}$ ± s, n=9)**

| Group | miR-93-5p | CyclinD1 | Cleaved-caspase-3 | Proliferation rate (%) | Apoptosis rate (%) |
|---|---|---|---|---|---|
| anti-miR-NC + si-LET | 1.00±0.12 | 0.80±0.08 | 0.46±0.05 | 100.02±10.25 | 10.24±1.02 |
| anti-miR-93-5p + si-LET | 0.36±0.03* | 0.45±0.04* | 0.84±0.09* | 68.24±6.80* | 24.24±2.40* |
| t | 15.522 | 11.739 | 11.073 | 7.751 | 16.106 |
| P | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |

Compared with anti-miR-NC + si-LET, *P<0.05.

**4. Discussion**

Through the loss of the ozonosphere the amount of UV radiation rises, the chance of skin lesions is a problem. In multiple skin disorders such as melanoma, hypertrophic scar and skin cell squamous carcinoma, lncRNAs are essential actors in epigenetics (Tang L. et al., 2020) (Li Z et al., 2019)(Chen L. et al., 2018) (Piipponen M. et al., 2020). In UV-induced HDF injury, the function of lncRNAs remains therefore to be explained.

In recent years lncRNA-LET has been shown to be correlating with many human diseases. A study indicates that lncRNA-LET is normally de-regulated in easily evident renal cell carcinoma and lncRNA-LET over-expression, which may cause cell cycle arrest, mitochondrial membrane disruption and

facilitate apoptosis, whereas the repression of lncRNA-LET is the opposite of findings (Ye Z. et coll., 2019). In addition, lNcRNA LET overexpression may even disrupt breast cancer and osteosarcoma cells' proliferation, infiltration and migration capacity (Zhou C.X. et al. 2018) (KOG, et al. 2018). Moreover, lncRNA-LET strengthens the heart and may minimize hypoxia-induced cardiomyocytic damage (Li Y., et al,2020) by overexpressing LNCRNA-LET. The function of lncRNA-LET in the UV in this analysis

Radiation-induced HDF injury has been studied, and lncRNA-LET expression has clearly increased in HDFs after UV radiation and could have a ratio of UV radiation-induced HDF wounds to lncRNA-LET. Furthermore, functional test findings revealed that intervention in lncRNA-LET expression greatly favored the proliferation and prevented the apoptosis of HDFs following UV radiation. Cyclin D1, a major protein for cell proliferation, promotes the transition from cell cycle to phase S by facilitating proliferation (Ding C, et al. 2020). The findings of functional assays indicate that the production of Cyclin D1 in HDFs after UV is observedly increased after intervention from the lncRNA-LET signal. In addition, there was increased expression of cleave-caspase 3, further evidence that lncRNA-LET expression interference may inhibit

apoptosis of HDFs following UV radiation. The findings described above suggest the up-regulation of the expression of lncRNA-LET is consistent with HDF radiation UV injury.

A recent study reported that the key regulatory mechanism of lncRNAs in the development and progression of skin diseases is to adsorb miRNAs to impede their expressions. For instance, lncRNA maternally expressed gene 3 (Meg3) interacts with miR-93-5p to modulate the expression of epiregulin and participate in UV-induced skin injury (Zhang N. et al., 2019). miR-93-5p was chosen as a possible target gene of lncRNA-LET in this research. Previous studies have shown that miR-93-5p, a tumor gene, has a carcinogenic impact on liver and cervical cancer and its overexpression promotes tumor formation and growth (Wang X. et al., 2018) (Sun XY, and others, 2019). The miR-93-5p thus stops the tumor cell distribution and movement of glioma (Wu H. et al. 2019). miR-93-5p is also able to prevent apoptosis of chondrocytes caused by lipopolysaccharide and has a safety function in the case of osteoarthritis (Sun Y. et al., 2020). In this analysis, the term miR-93-5p was found to be declining clearly following UV radiation in HDFs. The findings of the functional study have demonstrated that overexpressing miR-93-5p obviously increased Cycline D1's expression levels , reduced cleaved caspase-3 expression levels in HDFs after UV radiation, facilitated and suppressed apoptosis of hDFs after UV, consistent with interfering in UV-induced HDF I LET expression The findings of the double luciferase reporter test and RT-qPCR test both revealed that the lncRNA-LET was targeting and responding to miR-93-5p negatively. It was also discovered via a retrieval test that miR-93-5p inibition reverses the impact on the proliferation and apopdosis of HDFs after UV radiation from lncRNA-LET intervention partly. The previous findings indicate that lncRNA-LET plays a function in UV HDF accidents by targeting miR-93-5p. The following data are reported.

In summary, lncRNA-LET expression intervention can facilitate the proliferation and repression of HDF apoptosis after UV radiation by targeting and upgrading miR-93-5p. LncRNA-LET therefore is a promising and critical aim for the treatment of UV HDF damage.

**REFERENCES**

R., Shi H and others, (2018). Liu X. Protective effects of curcumin in human dermal fibroblasts against ultraviolet radiation-induced photoging. Med Rep Mol, 17(5), 7227-7237.

Greinert B, Henning S, et al. (2012) (2008). B. Double-strand splits of the UVA-induced DNA arise from repair of oxidative clustered damage to the DNA. Res, 40(20), 10263-10273 Nucleic acids.

Y, Xu Q, Y, et al. (2017). Large Noncoding RNA expression profiles in UVA-induced fibroblast human skins. Physiol Skin Pharmacol, 30(6), 315-323.

Y, Xu Q, Y, et al. (2017). Large Noncoding RNA expression profiles in UVA-induced fibroblast human skins. Physiol Skin Pharmacol, 30(6), 315-323.

Wang PL, Rosenberger B, Xia Y, et al. Long-standardized RNA-low tumor expression prevents invasion or metastasis through modulation of p53 development of esophageal squamous cell carcinoma. Med Rep. Mol, 13(4), 3074-3082.

Liu B, Pan CF, He ZC, and others (2016). Large non-coding RNA-LET suppresses lung adenocarcinoma tumour growth and EMT . Res Int, Biomed 2016(1), 4693471.

Wang Y et al. Zhang N, Zhong Z, et al. (2019). Efficient endogenous network analysis shows that the sponger miR-93-5p / epiregulin axis increases the lncRNA MEg3 's inflammatory

damage through UVB mediated murinous skin lesion. Ageing, 10664-10683, 11(22).

Liang Y, H Xie, et al. (2020). Tang L. Proliferative skin disorders, development and opportunities for long non-coding RNAs[D] in cutaneous biology. Prolif unit, 53(1), 12698.

Li, Tang, S, Tang, Tang. (2019). (2016). LncRNA intervention SPRY4-IT1 prevents melanoma cell growth, migration, and invasion by inactivating MAPK by upgrading miR-22-3p. Int J. Clin Exp Pathol, 12(2), 477-487.

Li Q, Li Q, and others. (2018). LncRNA Overexpression AC067945.2 The collagen production in skin fibroblasts is down-regulated and may be linked to the signalling pathways VEGF and Wnt. Biochem Cells, 45(2), 761-771. Cell Physiol.

Riihilä, et. (2020), Piipponen M, Nissinen L. P53-Regulated Long Noncoding RNA PRECSIT Facilitates Cutaneous Cell Carcinoma Development with STAT3 Signaling. In the J Pathol, 190.

Ye Z, DuanJ and others (2019). Wang L et al. LncRNA-LET prevents cell growth by controlling miR-373-3p in clear cell renal cell carcinoma. Cell Int, Lung, 19(1), 311.

Wang X, Yang N, etc. (2018). Zhou CX. In the treatment of breast cancer LncRNA LET acts as a tumor suppressor. Eur Rev Med Pharmacol Chem, 6002-6007, 22(18).

Kong G, Qi XJ, Wang JF. (2018). Effect of lncRNA LET on proliferation and invasion of osteosarcoma cells. Eur Rev Med Pharmacol Sci, 22(6), 1609-1614.

Li Y, Li J, Zhang P, et al. (2020). LncRNA-LET relieves hypoxia-induced injury in H9c2 cells through regulation of miR-138. J Cell Biochem, 121(1), 259-268.

Ding C, Wei R, Rodríguez RA, et al. (2019). LncRNA PCAT-1 plays an oncogenic role in epithelial ovarian cancer by modulating cyclinD1/CDK4 expression. Int J Clin Exp Pathol, 12(6), 2148-2156.

Wang X, Liao Z, Bai Z, et al. (2018). MiR-93-5p Promotes Cell Proliferation through Down-Regulating PPARGC1A in Hepatocellular Carcinoma Cells by Bioinformatics Analysis and Experimental Verification. Genes, 9(1), E51

Sun XY, Han XM, Zhao XL, et al. (2019). MiR-93-5p promotes cervical cancer progression by targeting THBS2/MMPS signal pathway. Eur Rev Med Pharmacol Sci, 23(12), 5113-5121.

Wu H, Liu L, Zhu JM. (2019). MiR-93-5p inhibited proliferation and metastasis of glioma cells by targeting MMP2. Eur Rev Med Pharmacol Sci, 23(21), 9517-9524.

Sun Y, Kang S, Pei S, et al. (2020). MiR93-5p inhibits chondrocyte apoptosis in osteoarthritis by targeting lncRNA CASC2. BMC Musculoskelet Disord, 21(1), 26.