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Mir-590-3p Regulates the Cardiomyocyte P19cl6 Proliferation, Apoptosis and Differentiation *In Vitro* by Targeting Ptpn1 via Jnk/Stat/Nf-Kb Pathway

Fanshun WANG [1], Anwar Ali JAMALI [2], Ashok Kumar LOHANO [2], Syed Shafqat Ali Shah RIZVI [3], Waqar Ahmed SABZOI [4], Hongqiang ZHANG [1] & Chunsheng WANG [1] *

[1] *Department of Cardiac Surgery, Zhongshan Hospital of Fudan University, Shanghai, 200000, China.*
[2] *Department of Medicine, People's University of Medical and Health Sciences for Women (PUMHSW), Nawabshah, Pakistan.*
[3] *College of Pharmacy, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, Pakistan.*
[4] *Department of Pharmaceutics, Faculty of Pharmacy, University of Sindh, Jamshoro, Pakistan*

***SUMMARY*. In our research, RT-qPCR was utilized to test the expression of miR-590-3p in cardiomyocyte P19CL6. Study was conducted at Department of Cardiac Surgery, Zhongshan Hospital of Fudan University, Shanghai, 200000, China, and study lasted for six months. MTT, EdU staining, caspase-3 activity detection, and flow cytometry experiments were adopted to test the influences of miR-590-3p on cell behaviors. Luciferase assay was utilized to confirm the binding situation of miR-590-3p and PTPN1. Western blot experiments were conducted to test the protein levels. Result showed that miR-590-3p was highly expressed in differentiated P19CL6. And silenced miR-590-3p suppressed proliferation but accelerated apoptosis of P19CL6. Moreover, PTPN1 was proved as the target of miR-590-3p. More importantly, overexpressed PTPN1 activated the JNK/STAT/NF-kB pathway and restrained differentiation of P19CL6. It was concluded that miR-590-3p could regulate the cardiomyocyte P19CL6 proliferation, apoptosis, and differentiation *in vitro* by targeting PTPN1 via JNK/STAT/NF-kB pathway.**

RESUMEN. En esta investigación se utilizó RT-qPCR para evaluar la expresión de miR-590-3p en el cardiomiocito P19CL6. El estudio se realizó en el Departamento de Cirugía Cardíaca, Hospital Zhongshan de la Universidad de Fudan, Shanghai, 200000, China, y el estudio duró seis meses. Se adoptaron MTT, tinción con EdU, detección de actividad de caspasa-3 y experimentos de citometría de flujo para evaluar las influencias de miR-590-3p en los comportamientos celulares. El ensayo de luciferasa se utilizó para confirmar la situación de unión de miR-590-3p y PTPN1. Se realizaron experimentos de Western blot para probar los niveles de proteína. El resultado mostró que miR-590-3p se expresó altamente en P19CL6 diferenciado. Y miR-590-3p silenciado suprimió la proliferación pero aceleró la apoptosis de P19CL6. Además, PTPN1 se demostró como el objetivo de miR-590-3p. Más importante aún, la PTPN1 sobreexpresada activó la vía JNK/STAT/NF-kB y restringió la diferenciación de P19CL6. Se concluyó que miR-590-3p podría regular la proliferación, apoptosis y diferenciación de cardiomiocitos P19CL6 *in vitro* al dirigir PTPN1 a través de la ruta JNK/STAT/NF-kB.

KEY WORDS: JNK/STAT/NF-kB pathway, MiR-590-3p, P19CL6, PTPN1.

* Author to whom correspondence should be addressed. *E-mail:* washington2255@yandex.com

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