Ruprecht-Karls-Universität Heidelberg
Medizinische Fakultät Mannheim
Dissertations-Kurzfassung

MicroRNA-30b-5p Regulates the Pericyte-like Differentiation of Adipose-derived Stem cells and the Phenotypic Switch of Human Aortic Vascular Smooth Muscle Cells

Autor: Ziwei Zeng
Institut / Klinik: Chirurgische Klinik
Doktorvater: Prof. Dr. N. Rahbari

Ischemic diseases are a type of diseases with high morbidity and mortality worldwide. Therapeutic angiogenesis, by restoring adequate circulation in the ischemic area, rescuing dying cells, promoting tissue regeneration and functional restoration in the ischemic area, and alleviating or even preventing organ failure, is the leading research direction for the current treatment of ischemic diseases. Pericytes are the essential cell in the formation of new vessels. Adipose-derived stem cells (ADSC) can differentiation into pericytes. To understand the underlying mechanism of the pericytes-like differentiation of ADSC can provide potential treatment targets of ischemic diseases. Here, we established this study to explore the function of miR-30b-5p in pericytes-like differentiation of ADSC. In this study, we found that miR-30b-5p is overexpression when ADSC differentiation into pericytes. After the ADSCs were transfected with miR-30b-5p, the process of pericytes-like differentiation of ADSC can be promoted, while anti-miR-30b-5p can inhibit ADSC differentiation into pericytes. The mRNA expression level of AP3S1 and GOLGA1 was regulated by miR-30b-5p. Then, after the ADSCs were transfected with si-AP3S1 and si-GOLGA1, the process of pericytes-like differentiation of ADSC can also be enhanced. Furthermore, the inhibition of pericytes-like differentiation of ADSC caused by anti-miR-30b-5p can be reversed by transfected with si-AP3S1 and si-GOLGA1. Our data support that miR-30b-5p regulates pericytes-like differentiation of ADSC. In the further study, we will explore the underlying mechanism of AP3S1 and GOLGA1 to find more potential therapeutic methods to enhance ADSC differentiation into pericytes to improve ischemic diseases.
MicroRNA-30b-5p Regulates the Phenotypic Switch of Human Aortic Vascular Smooth Muscle Cells
Peripheral artery disease (PAD) is a disease of abnormal narrowing and obstruction of arteries, which most commonly influence the blood supply of the legs. Stent angioplasty is an important treatment for PAD. However, in-stent restenosis (ISR) may casue treatemtn failure and even re-occusion of the blood vessel. Therefore, finding a way to prevent ISR is clinically urgent and essential. Based on published studies, miR-30b-5p is overexpression in ISR pateints, and vascular smooth muscle cells (VSMCs) play a key role in the development of ISR. Unfortuanetly, whether miR-30b-50 regulates VSMCs to cause ISR is still eclusive. Therefore, we conducted this study aimed to explore the function of miR-30b-5p in VSMCs. In our study, we found that miR-30b-5p is overexpression during the VSMC switch from synthetic type to contractile type. After the VSMCs were transfected with miR-30b-5p, the VSMC keeps a contractile type, while the VSMC keeps a synthetic type after being transfected with anti-miR-30b-5p. The mRNA expression level of GOLGA1 and SCAMP1 was regulated by miR-30b-5p. After the VSMCs were transfected with si-GOLGA1 and si-SCAMP1, the VSMC showed a similar phenotype compared with the VSMC transfected with miR-30b-5p. At the same time, the VSMC switching from contractile type to synthetic type, caused by transfected with anti-miR-30b-5p, can be inhibited by transfected with si-GOLGA1 and si-SCAMP1. Based on our results, it can be speculated that miR-30b-5p regulates the phenotypic switch of VSMC by targeting GOLGA1 and SCAMP1.