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Role of transient receptor potential canonical channel 6 (TRPC6) in diabetic kidney disease by regulating podocyte actin cytoskeleton rearrangement

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Abstract

Podocyte injury is an important pathogenesis step causing proteinuric kidney diseases such as diabetic kidney disease (DKD). Actin cytoskeleton rearrangement in podocyte induced by multiple pathogenic factors is believed the key process resulting into its injury. Many studies have recently shown that transient receptor potential canonical channel 6 (TRPC6) in podocyte plays a critical role in the development and progression of proteinuric kidney disease, that participate in proteinuria formation by regulating its actin cytoskeleton rearrangement. This review aims to summarize the TRPC6 role in podocyte on DKD by regulating the actin cytoskeleton rearrangement, thereby help further broaden our views and understanding on the mechanism of DKD and provide a theoretic basis for exploring new therapeutic targets for DKD patients.

Biography:

Ping Li is the chairman of Beijing Key Lab for Immune-Mediated Inflammatory Diseases, Institute of Clinical Medical Science, China-Japan Friendship Hospital. She has engaged in the clinical and basic research of integrated traditional Chinese and western medicine treating kidney disease for 36 years and successively presided over and undertook more than 20 national and provincial level scientific research projects. She has won the second prize of national science and technology progress award and 11 prizes above provincial and ministerial level as the first accomplisher. She has obtained 5 national invention patents. She has published more than 200 papers in journals and academic conferences at home and abroad, of which more than 80 papers are included in SCI. She wrote 11 academic works and edited 5 academic works

Speaker Publications:

1. Tangshen formula alleviates hepatic steatosis by inducing autophagy through the AMPK/SIRT1 pathway
2. Role of transient receptor potential canonical channel 6 (TRPC6) in diabetic kidney disease by regulating podocyte actin cytoskeleton rearrangement

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