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사구체 손상에서 MYH9의 역할

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MYH9 in Podocytes: A New Player in Health and Disease

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Aim: MYH9 is a podocyte-expressed gene encoding a nonmuscle myosin IIA heavy chain and plays a role in several important cellular functions, including cytokinesis, cell motility and maintenance of cell shape. Despite several groups have investigated extensively to understand the role and function of MYH9 in glomerular disease using animal models, little is known about the glomerular-related functions of MYH9. Here, we used the immortalized mouse podocyte in order to better understand the relationship between MYH9 and podocyte injury.
Methods: We knocked down MYH9 gene by target siRNA in conditionally immortalized mouse podocytes. MYH9 siRNA-transfected podocytes were treated with angiotensin II (Ang II), a key mediator of various kidney injury, for 72h. Podocyte cytoskeleton, migration, adhesion, and permeability were assessed. Gene and protein expression were examined by real-time RT-PCR, Western blot and immunofluorescence.
Results: Ang II stimulation induced decreased expression of MYH9, as well as synaptopodin and nephrin in differentiated podocytes. Knockdown of MYH9 resulted in morphological changes by reorganization of actin cytoskeleton, reduced cell adhesion, and increased cell migration and albumin leakage through glomerular filteration barrier than cells expressing MYH9. MYH deleted-podocytes were more susceptible to induce those defects under Ang II stimulation. b-1 integrin, podocyte-specific cell adhesion receptor and a-actinin-4, a key component of actin cytoskeleton were reduced in MYH9-depleted podocytes.
Conclusion: These results suggest that MYH9 is a key component to maintain the biological function of podocytes. Its impairment may cause actin cytoskeleton rearrangement and alteration of functional integrity of podocytes, which might finally lead to an impaired glomerular filtration barrier and proteinuria.
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