Genetics and Molecular Research

Preliminary *in vitro* analysis of mechanism of cardiac microvascular endothelial barrier function

W. Dong[1]*, G.N. Zhang[2]* and S.H. Gao[3]

[1]Intensive Care Unit, Jining No. 1 People's Hospital, Jining, Shandong Province, China
[2]Department of Endocrinology, Jining No. 1 People's Hospital, Jining, Shandong Province, China
[3]Department of Nursing, Jining No. 1 People's Hospital, Jining, Shandong Province, China

*These authors contributed equally to this study.
Corresponding author: S.H. Gao
E-mail: gaoshuhongdn@163.com

Genet. Mol. Res. 15 (4): gmr15048864
Received June 8, 2016
Accepted August 30, 2016
Published October 17, 2016
DOI http://dx.doi.org/10.4238/gmr15048864

ABSTRACT. To preliminarily clarify the mechanism of cardiac microvascular endothelial barrier function leading to heart failure, primary HMVEC-D cells were selected and cultured for amplification. The cells were infected with adenovirus vector containing the ADP-ribosylation factor 6 (*Arf6*) Q67L gene. Full-length and functional fragments of myeloid differentiation primary response 88 and ARF nucleotide-binding site opener genes were established and transfected into HEK293T cells. GTP-Arf6 pull-down experiment, fluorescent quantitative real-time PCR, immuno-coprecipitation, and transendothelial electrical resistance analysis were conducted.

Interleukin-1β (IL-1β) induced increase in vascular permeability, whereas inhibitor SC514 blocked IL-1β-induced transfer of nuclear factor-κB into the nucleus, from the cytoplasm. Increase in amount of activated Arf6 promoted reduction in transendothelial electrical resistance. In addition, SecinH3 significantly inhibited increase in vascular permeability, and the progression of heart failure was significantly relieved. Cardiac microvascular endothelial barrier function can lead to heart failure. However, IL-1β induced increase in vascular permeability, which nullified the function of cardiac microvascular endothelial barrier. These findings are closely related to the activation of the Arf6-VE-cadherin signaling pathway.

Key words: Heart failure; Endothelial barrier function; IL-1β; SC514; Arf