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Activation of the ERK1/2 pathway by the CaMEK gene via adeno-associated virus serotype 9 in cardiomyocytes

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ABSTRACT. Extracellular signal-regulated kinase (ERK1/2) is one of the mitogen-activated protein kinases, key components of the reperfusion injury salvage kinase pathway, which plays an important role in protecting the myocardium from lethal ischemia-reperfusion injury. Constitutive activation of the mitogen-activated protein kinase kinase 1 (CaMEK) can promote ERK1/2 expression, which is thereby expected to exert protective action on the heart against ischemia-reperfusion injury. The adeno-associated virus serotype 9 vector (AVV9) is a novel tool for gene therapies targeting human diseases owing to its nonpathogenic capability for transducing nondividing cells and its long-term transgene expression. We used a recombinant AAV9 vector to deliver the CaMEK gene into cardiomyocytes and assessed whether AAV9 vector-mediated CaMEK gene transfection could enhance the long-term expression and activity of ERK1/2. Our observations suggest that AAV9-mediated gene expression is preferentially restricted

to cardiomyocytes and that mediated CaMEK gene transfection enhanced the expression of ERK1/2 phosphorylation and consequently upregulated the expression of downstream components of ERK1/2 and its transcription factors.

Key words: Recombinant adeno-associated virus serotype 9; Constitutive activation of the mitogen-activated protein kinase kinase 1; Extracellular signal-regulated kinase

