and stable knock down of STAT3. Treatment of cardiomyocytes with shERK resulted in ERK1/2 phosphorylation and a decrease in the expression of CAR accompanied by the elevation of LDH levels in infected with CVB₃ cells.

Conclusions In this study we investigated the effect of signalling through the Raf-MEK-ERK1/2 pathway on CAR expression in cardiac myocytes that are potential targets for adenovirus-based therapies. Our findings in modulation of CAR expression, may lead to new strategies in the gene therapy of DCM.

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THE CROSSTALK BETWEEN ERK1/2 AND STAT3 IN THE REGULATION OF CAR EXPRESSION DURING CVB3 INFECTION IN CARDIOMYOCYTE.

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Objectives We performed a novel analysis of cardiac expression of receptors for several adenovirus serotypes with a focus on expression of CAR, as adenoviruses targeting receptor has been used in various applications, that more than the viral receptor which is significantly induced in the heart tissue of CVB₃ infected and considered to be the dominant aetiology of viral myocarditis.

Methods We treated cardiomyocytes with culture medium in the uninfected or infected of CVB₃ (MOI=100). The lentiviral vector system derived from HIV-1 was used to express short hairpin RNAs (shRNA) directed against STAT₃ and ERK1/2 activation was blocked with U0126, an ERK1/2 inhibitor. Western blot was used to observe the level of CAR, ERK and STAT₃. The degrees of cells injury were judged by LDH levels in cells supernatant.

Results Up-regulation activities of ERK1/2 after CVB₃ infected with cardiomyocytes, accompanied by positive correlation of the expression of CAR. Treatment of cardiomyocytes with Pharmacological inhibition of ERK1/2 phosphorylation with U0126 resulted in a dramatic increase in the expression of CAR. U0126 induced the JNK/STAT₃ pathway activity to prevent cells from injury. Lentivector-based short hairpin RNAs provide efficient