



## Letter to the Editor

## LncRNA GAS5 aggravates pathological cardiac hypertrophy by targeting miR-135b to mediate apoptosis

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Dear Editor,

Recently, Chu et al. reported that miR-135b was significantly down-regulated in pathological cardiac hypertrophy, and its overexpression could attenuate cardiac hypertrophy, probably mainly by down-regulating the hypertrophic marker genes to attenuate hypertrophic signals [1], which demonstrated that miR-135b may play a crucial role in cardiac hypertrophy. However, the underlying molecular mechanisms regulating miR-135b expression remain largely unclear.

LncRNA growth arrest specific transcript 5 (GAS5) has been widely recognized as a known tumor suppressor, but it exerts crucial functions in regulating cell apoptosis and proliferation in both cancerous cells and noncancerous cells. Recently, Yue et al. [2] demonstrated that GAS5 down-regulation could ameliorate palmitic acid-induced myocardial inflammatory injury via regulating miR-26a. Moreover, a recent study has confirmed that lncRNA GAS5 silence could protect cardiomyocytes H9c2 cells from hypoxia-induced cell damage by inhibiting apoptosis through sponging miR-142-5p [3]. Therefore, it is reasonable for us to speculate that GAS5 may be a new therapeutic target for cardiac pathological hypertrophy by interacting with vital miRNAs. Notably, Hu et al. [4] showed that inhibition of miR-135b could promote cell apoptosis,

indicating that miR-135b is a key regulator of apoptosis. It is noteworthy that GAS5 could directly bind to miR-135b and negatively regulate its expression as a competing endogenous RNA [5].

Hence, we speculated that GAS5 may aggravate pathological cardiac hypertrophy by targeting miR-135b to promote cardiomyocyte apoptosis. However, this speculation needs to be further proved by more systematic experiments.

## Conflicts of interest disclosures

None disclose.

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