Non-coding RNAs and bronchial smooth muscle hyperresponsiveness in allergic bronchial asthma

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Non-coding RNAs, such as microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), play important roles in normal and diseased cell functions. A small GTPase RhoA is a key protein of bronchial smooth muscle (BSM) contraction, and an up-regulation of RhoA has been demonstrated in BSMs of experimental asthma. Our previous study also demonstrated that RhoA translation was controlled by a miRNA, miR-133a, in BSMs. In human BSM cells (hBSMCs), an up-regulation of RhoA was observed when the function of endogenous miR-133a was inhibited by its antagomir. Treatment of hBSMCs with interleukin-13 (IL-13) caused an up-regulation of RhoA and a down-regulation of miR-133a. In a murine experimental asthma, increased expression of IL-13 and RhoA and the BSM hyperresponsiveness were observed. Interestingly, the level of miR-133a was significantly decreased in BSMs of the diseased animals. These findings suggest that RhoA expression is negatively regulated by miR-133a in BSMs, and that the miR-133a down-regulation causes an up-regulation of RhoA, resulting in an augmentation of the contraction. Recent studies also revealed an inhibitory effect of lncRNA *Malat1* on the miR-133a function. Thus, lncRNAs/miRNAs might be key regulators of BSM hyperresponsiveness, and provide us a new insight into the treatment of airway hyperresponsiveness in asthmatics.