Promotion of Apoptosis Does Not Necessarily Mean Inhibition of Remodeling

To the Editor:

Hypoxic pulmonary hypertension is a serious disease characterized by hypoxic pulmonary vasoconstriction and pulmonary vascular remodeling. In the article “The MicroRNA-328 Regulates Hypoxic Pulmonary Hypertension by Targeting at Insulin Growth Factor 1 Receptor and L-Type Calcium Channel-1C,” the authors have drawn a conclusion that miR-328 is downregulated in pulmonary arteries from hypoxic rats and patients with pulmonary hypertension, affecting hypoxic pulmonary vasoconstriction and pulmonary vascular remodeling by targeting Cav1.2 and insulin-like growth factor 1 receptor, leading to hypoxic pulmonary hypertension. However, from the data presented, the authors claimed only that miR-328 induced pulmonary artery smooth muscle cell (PASMC) apoptosis through repression of insulin-like growth factor 1 receptor. Promoting PASMC apoptosis does not necessarily mean inhibiting pulmonary vascular remodeling. The authors stated only that miR-328 inhibited pulmonary artery remodeling related to PASMC apoptosis. The effects of miR-328 on pulmonary arteries remodeling and PASMC apoptosis may be 2 accompanying phenomena. In addition, the relationship between pulmonary artery remodeling and PASMC apoptosis was confusing to the reader. In addition, the authors seemed to measure apoptosis according to the results of 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT). Although the apoptosis assay is closely related to MTT measurement, we do not think that the MTT assay can be used to analyze apoptosis. MTT assay measures the rate of surviving cells and cannot distinguish the manner and stages of cell death. Reduction in MTT assay result from cell death and/or inhibition of proliferation; however, cell apoptosis is only one type of cell death. In addition, early stage apoptotic cells may be recognized as normal cells according to the MTT measurement. Therefore, it did not seem appropriate to measure apoptosis by MTT. In addition, the role of Cav1.2 in miR-328–mediated inhibition of hypoxic pulmonary vasoconstriction and the role of insulin-like growth factor 1 receptor in miR-328–induced PASMC apoptosis lack direct evidence. The authors only cited references. As we stated previously, miR-328–induced apoptosis and miR-328–inhibited insulin-like growth factor 1 receptor may be 2 accompanying phenomena. With regard to the authors’ claim that miR-328 induces PASMC apoptosis through repression of insulin-like growth factor 1 receptor, we suggest that additional experiments aimed at determining whether overexpression of insulin-like growth factor 1 receptor can blunt the stimulating effect of miR-328 on PASMCs apoptosis be carried out.

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Disclosures

None.

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