The transcription factor myocyte enhancer factor 2 (MEF2) is significantly impaired in pulmonary arterial hypertension (PAH).

Inhibition of class IIa HDACs restored MEF2 activity in pulmonary arterial endothelial cells (PAECs), as demonstrated by increased expression of targets, including miR-424 and miR-503.

Augmentation of MEF2 activity holds a potential therapeutic value in PAH.

Selective HDAC IIa inhibition was identified as a viable alternative approach to avoid the potential adverse effects of broad spectrum HDAC inhibition in PAH.