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For Linux, Windows And Mac 5.12 MB \]. Consistent with this, our preliminary data also indicated that the expression of miR-31 was increased in the PASMCS of the PAH rats and was inversely correlated with the mRNA expression of UCP2, a hallmark of mitochondrial uncoupling \[[20](#sct312784-bib-0020){ref-type="ref"}, [21](#sct312784-bib-0021){ref-type="ref"}, [22](#sct312784-bib-0022){ref-type="ref"}\]. Our findings are consistent with the previous studies which showed that repressing miR-31 in cancer stem cell increased their mitochondrial

uncoupling, causing a severe reduction in stem cell self-renewal and tumorigenesis \[[11](#sct312784-bib-0011){ref-type="ref"}, [12](#sct312784-bib-0012){ref-type="ref"}, [23](#sct312784-bib-0023){ref-type="ref"}\]. Our data indicate that the effects of miR-31 on promoting proliferation and reducing apoptosis could be attenuated by increasing UCP2 in PAH rat PSMCs. However, how UCP2 is involved in the cellular effects of miR-31 in PAH is still not fully understood. In terms of how these two molecules are connected, we found that the expression of miR-31 was

decreased while UCP2 was increased after overexpressing miR-31 in PASMCs, which could lead to mitochondrial uncoupling. Further experiments indicated that inhibiting UCP2 in PASMCs could recover the proliferation and apoptosis phenotypes which were induced by miR-31 overexpression in PAH rat PASMCs. These data confirmed our hypothesis that miR-31-mediated mitochondrial uncoupling could be involved in the proliferation and apoptosis of PAH PASMCs. Our data indicated that the overexpression of UCP2 rescued the mitochondrial uncoupling effect of miR-31 on P

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