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MiR-200a-3p Accelerated Hypoxia/Reoxygenation Injury in HCM Cells by Enhancing IGF2R via Wnt/β-catenin Signalling Pathway

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ABSTRACT The present study examined functions of miR-200a-3p accelerated progressions of HCM cells via IGF2R and Wnt/β-catenin signalling pathway after hypoxia/reoxygenation treatment in vitro. CCK-8 showed that cell viability of HCM was inhibited while apoptosis rates detected by flow cytometry were promoted in a time dependent manner after H/R (12 hours and 24 hours). Beyond that, Bcl-2 and c-IAP1 were decreased but Bax and caspase-3 were upregulated by H/R treatment. IL-1β, IL-6, TNF-α and NLRP3 were also increased after treatment. RT-qPCR showed increased expressions of miR-200a-3p by H/R treatment while its inhibitor elevated cell viability but depressed apoptosis rate and pro-inflammatory cytokines' expressions. IGF2R was upregulated after H/R treatment and its downregulation magnified effects of suppressed miR-200a-3p. HIF-1α/Wnt/β-catenin signalling pathway was activated by miR-200a-3p and IGF2R while IWP-2 treatment abolished the activation of Wnt3a and β-catenin, causing decreased apoptosis and pro-inflammatory cytokines' expressions but accelerated the cell viability.