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LncRNA HOTAIR/MiR-217/GPD2 Axis Medicates Hypoxia Injury of Myocardial Cells

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ABSTRACT This study examined impacts of lncRNA HOX Transcript Antisense RNA (HOTAIR), miR-217 and Glycerol-3-phosphate dehydrogenase 2 (GPD2) on progressions of cardiomyocytes after hypoxia damage. Hypoxia treatment induced low cell viability and increased apoptosis. RT-qPCR evaluated suppressed expressions of lncRNA HOTAIR. Overexpressed lncRNA HOTAIR accelerated AC16 cell viability but restrained cell apoptosis and pro-inflammatory protein expressions while the knockdown of HOTAIR caused opposite results. MiR-217 then was examined to be inhibited by HOTAIR overexpression, whose upregulation reduced AC16 cell viability but facilitated apoptosis and pro-inflammatory protein expressions. Luciferase reporter test then verified that GPD2 was bound and decreased by miR-217, which promoted AC16 cell viability but hampered cell apoptosis and pro-inflammatory protein expressions after overexpression. Moreover, PI3K/AKT signaling pathway was activated by overexpression of GPD2.