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**Title:** Adiponectin may inhibit chronic intermittent hypoxia-induced endoplasmic reticulum stress and cell apoptosis in genioglossus

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**Body:** Objective To investigate the effects of chronic intermittent hypoxia (CIH) and adiponectin (Ad) supplement on endoplasmic reticulum stress (ERS) and cell apoptosis in genioglossus. Methods Wistar rats were randomly divided into three groups: normal control group (group A), CIH group (group B) and CIH+Ad group (group C) with 10 rats in each. Rats in group A were kept breathing normal air, while rats in both group B and group C received same CIH environment. However, rats in group C was given intravenous Ad supplement. At the end of experiment (day 35), the expression of glucose-regulated protein 78 (GRP78), P38, c-Jun NH-terminal kinase (JNK) and C/EBP homologous protein (CHOP) of genioglossus were tested with western blot assay. The cell apoptosis was measured through TUNEL and Annexin V/PI staining. Results Compared with control group, in CIH group the P38 was significantly activated, the expression of GRP78 and CHOP proteins of genioglossus were significantly upregulated, and the rate of cell apoptosis was significantly elevated (all  $P < 0.01$ ). However, compared with CIH group, in group C the activation of P38 was partially inhibited, the expression of GRP78 and CHOP proteins was significantly weakened, and the rate of cell apoptosis was remarkably lower (all  $P < 0.05$ ). There was no significant difference of JNK expression of genioglossus among three groups ( $P > 0.05$ ). Conclusions CIH could induce ERS and significantly enhance cell apoptosis through activation of P38-CHOP pathway in genioglossus. Supplement of adiponectin could attenuate excessive ERS, inhibit activation of P38-CHOP pathway, and thus further reduce the rate of cell apoptosis in genioglossus.