論文摘要範本

Genome-wide gene expression microarray identifies novel genes related to endothelial tight junctions and apoptosis in patients with obstructive sleep apnea

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Purpose: We aim to identify novel molecular mechanisms by which intermittent hypoxia with re-oxygenation (IHR) leads to adverse consequences, such as hypertension and excessive daytime sleepiness (EDS) in patients with obstructive sleep apnea (OSA).

Materials and Methods: We analyzed whole-genome gene expression profiles of peripheral blood mononuclear cells from 48 patients with sleep-disordered breathing stratified into four groups: primary snoring (PS), moderate to severe OSA (MSO), very severe OSA (VSO), and very severe OSA patients with long-term continuous positive airway pressure (CPAP) treatment (VSOC).

Results: Comparisons of the microarray gene expression data identified eight genes up-regulated with OSA and down -regulated with CPAP treatment, and five genes down-regulated with OSA and up-regulated with CPAP treatment (all p values<0.05). Protein expression levels of 2 genes related to endothelial tight junctions (AMOT P130, and PLEKHH3; both p values <0.001), and 3 genes related to anti-or pro-apoptosis (ADAR1 P150, BIRC3, and GALIG; all p values<0.05) were all increased in the VSO group, while AMOT P130 was further increased, and PLEKHH3, BIRC3, and ADAR1 P150 were all decreased in the VSOC group. AMOT P130 protein expression was increased in OSA patients with EDS, BIRC3 protein expression was decreased in OSA patients with hypertension, and GALIG protein expression was increased in OSA patients with chronic kidney disease (CKD). In vitro IHR (7 cycles/day, 4 days) stimuli resulted in over-expression of ADAR1 P150.

Conclusions: We identified a novel association between AMOT/PLEKHH3/BIRC3/ADAR1/GALIG over-expressions and high Apnea-Hyponea Index in OSA patients. AMOT and GALIG may constitute an important determinant for the development of EDS and CKD, respectively, while BIRC3 may play a protective role in the development of hypertension in OSA.

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