**投稿論文格式與規範**

1. 摘要以**英文繕寫**，內容以**一頁為限**，未依照規定的格式，或不符直接製版印刷之條件者，將逕予退稿。
2. 稿件需為Microsoft Word電子檔案，文稿採用A4規格 (297 mm × 210 mm)。版面設定部分，上下左右邊界**均為 2 cm**。
3. 內文由左至右橫式書寫，中文字體為**標楷體**，英文字體為**Times New Roman**，單行間距。中英文題目字體大小皆為 12級、粗體；中英文作者與單位名稱字體大小皆為11級；英文摘要內文字體大小為12級。數字與英文皆為半形。**演講者為第一作者並請加底線**，英文姓名請以**全名書寫**(Meng-Chih Lin)，全文請適當分段。
4. 請勾選投稿類別A與B (原著論文或病例報告論文；口頭報告或海報競賽)。

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| A. | ■原著論文 (Original Paper) | □病例報告論文 (Case Report) |
| B. | □口頭報告 ( Oral Presentation) | ■海報競賽 (Post) |

**全基因體微矩陣基因表現全貌辨識出調控阻塞性睡眠呼吸中止症的新分子機轉: 內皮細胞緊密接合和細胞凋零**

**範例**

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**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.