KSN SEOUL, KOREA 2023
April 27(Thu) - 30(Sun), 2023
Coex, Seoul, Korea

Abstract Type : Poster
Abstract Submission No. : 1741

Shortening of the primary cilia length involves urine concentration in the kidneys

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Objectives: The primary cilium, a microtubule-based cellular organelle, acts as a mechano-sensor for monitoring the fluid flow as well as various biological functions in cells. In kidneys, the primary cilia protrude into the tubular lumen and directly contact pro-urine flow and components. However, its role on urine concentration remains to be defined. Here, we investigated the association of primary cilia with water balance and urine concentration.

Methods: Mice were freely accessed to either water for normal water intake (NWI) and not allowed to access to water for water deprivation (WD). Some mice were administered tubastatin, an inhibitor of histone deacetylase 6 (HDAC6), which regulates the acetylation of α-tubulin, a core protein of microtubule.

Results: WD induced a reduction of urine output and increase of urine osmolarity, along with apical plasma membrane localization of AQP2. WD shortened cilia in tubule cells along with increased HDAC6 activity. After WD, the expression of acetylation α-tubulin, which is composed of microtubule cilia and regulated by HDAC6, decreased without decreased total α-tubulin expression in the kidney. Tubastatin prevented the shortening of cilia and the increase of HDAC6 activity along with increased acetylated α-tubulin expression. Also, tubastatin prevented WD-induced reduction of urine output, increase of urine osmolality, and apical plasma membrane localization of AQP2.

Conclusions: Change in water depletion shortens primary cilia length through HDAC6 activation and α-tubulin acetylation, and HDAC6 inhibition blocks changes in cilia length and urine output. This suggests that cilia length alteration involves, at least in part, the regulation of body balance.