

# KSN 2016 Abstract Submission

## *CKD & associated complications*

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**Diabetic condition is associated with decreased  $\alpha$ klotho expression and re-localization of sodium-phosphorous cotransporter (Napi2a/2c) in the kidney.**

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**Background:** Diabetic nephropathy (DN) is the leading cause of the chronic kidney disease (CKD) all over the world.  $\alpha$ -klotho, as an antiaging protein, is mainly expressed in the kidney.  $\alpha$ -klotho is a major regulator of mineral metabolism and is deranged in the CKD. Here, we explored  $\alpha$ klotho expression in diabetic condition (*in vivo* and *in vitro*), and how this change in  $\alpha$ klotho could affect phosphorus metabolism.

**Methods:** We extracted the kidneys from 15-week-old mice (C57BL6 db/db) and analyzed  $\alpha$ klotho expression via immunohistochemistry assay. db/m mice were used as control. In addition, we analyzed  $\alpha$ -klotho and sodium-phosphorous cotransporter (Napi2a/2c) expression from S1 proximal tubule cells (immortalized) of the wild-type C57BL6 mice after treatment with normal glucose (5mM, NG) or high glucose (30mM, HG) by confocal microscopy.

**Results:** Immunohistochemistry exhibited significantly decreased expression of  $\alpha$ -klotho from db/db mouse kidney, as compared with control. *In vitro*, HG treated cells expressed remarkably lower level of  $\alpha$ klotho. HG treatment caused re-localization of Napi2a/2c molecules toward the cell membrane, which is the main action site of sodium-phosphorus cotransporter.

**Conclusion:** Our results indicate that renal  $\alpha$ -klotho decreases in diabetic conditions. Napi2a/2c was re-localized to the cell membrane after high glucose treatment. Our current study will continue to seek for mechanistic links between high glucose,  $\alpha$ klotho and renal phosphorus regulation.

**Table:**

**Keywords:**  $\alpha$ klotho, diabetic nephropathy, phosphorus metabolism