GRAPHICAL ABSTRACT SAMPLE WORK

Restoration of branched-chain amino acid catabolism improves kidney function in preclinical cardiovascular-kidney-metabolic syndrome models

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Restoration of branched chain amino acid catabolism improves kidney function in preclinical cardiovascular- kidney-metabolic syndrome models

PUBRICA Knowledge Works

Methods: Multiple pre-clinical CKM models



ZSF1 Obese Rat



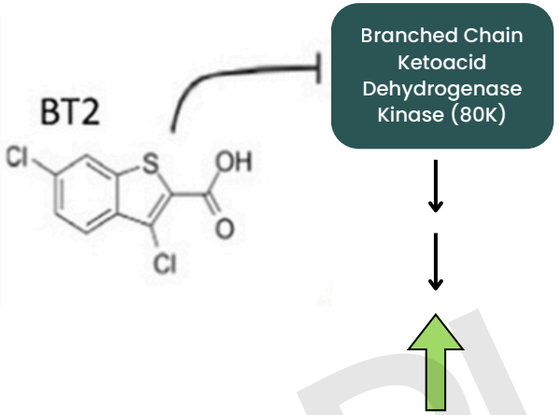
SDT Unx Rat + High Salt Water

Multi-omics profiling

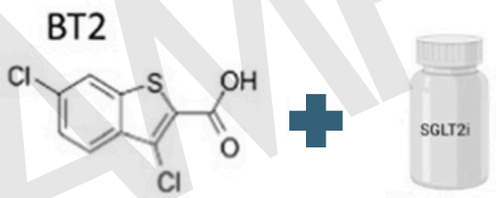
BCAAS →CKM

Dysfunctional BCAA Breakdown

Intervention: BT2 alone or in combination with SGLT2i

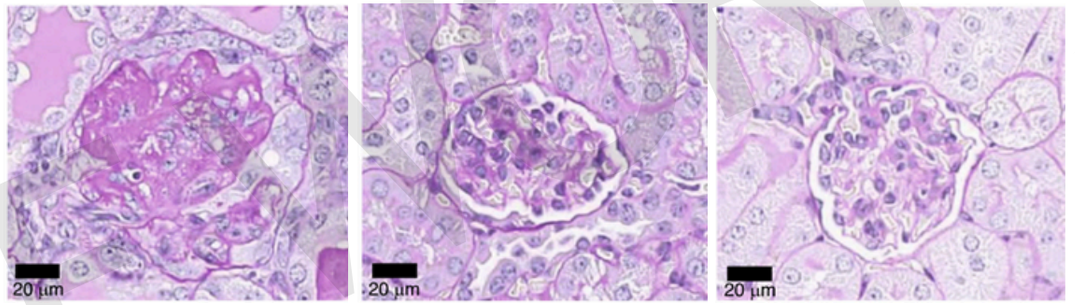


BT2 monotherapy



BT2 + SGLT2i combination therapy

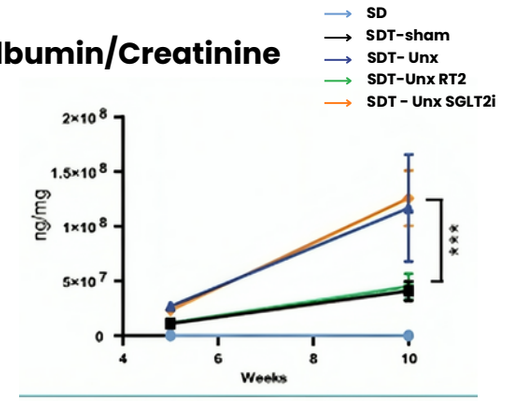
Results: BT2 monotherapy and combination with SGLT2i improved CKM parameters.

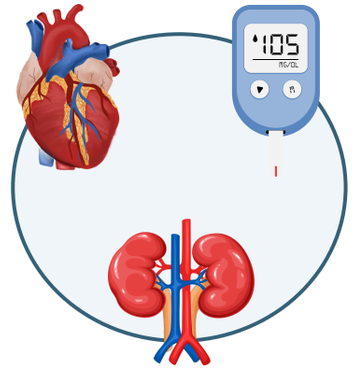


Kidney mitochondrial density/function

Urine protein content, kidney hypertrophy, and kidney pathological damages

Albumin/Creatinine





Improved CKM

SAMPLE WORK

Image Adapted from Bollinger E, Williams G, Piper ME, et al. Restoration of branched-chain amino acid catabolism improves kidney function in preclinical cardiovascular-kidney-metabolic syndrome models. Kidney Int. 2025;108(2):310-316. doi:10.1016/j.kint.2025.04.025

CONCLUSION: Improving BCAA catabolic defects with BT2, a BDK inhibitor, can ameliorate CKM syndrome. Furthermore, BDK inihbitors may show additional benefit when co-administered with SGLT2 inhibitors.