

Contributions of obesity to kidney health and disease — insights from mendelian randomisation and the human kidney transcriptomics

Kidney transcriptome profiling and pathway analyses.

We collected demographic and clinical information matching data on up to 467 human kidney tissue samples drawn from five studies of the Human Renal Tissue Resource. The tissue samples were taken either from the unaffected pole of the kidneys surgically removed due to cancer or from pre-implementation biopsies conducted prior to kidney transplantation. Gene expression was quantified in units of transcripts per million (TPM) by kallisto from poly-A selected Illumina libraries (mean: 32 million paired reads per sample). Our quality control process selected renal genes for further analysis – their expression values were log-transformed, normalised by the robust quantile method and standardised by the rank-based inverse normal transformation as previously reported . BMI was calculated based on weight and height as reported before WC was measured using a measuring tape placed around the trunk at the midline level. Hypertension was defined as BP values $\geq 140/90$ mmHg on at least two separate occasions and/or being on pharmacological antihypertensive treatment or collected from hospital documentation by the recruitment team. Diabetes was defined as either a self-reported history of diabetes and/or being on hypoglycaemic medications or was reported in hospital documentation at the time of recruitment.

Table S5. Demographic and clinical information for 467 samples from the Human Renal Tissue resource. Values reported are either means and standard deviations (in parentheses) or counts and percentages (in parentheses). Waist circumference was available for 354 subjects.						
Variable summary						
Number	467					
Age (years)	57.6 (13.9)					
Male sex	293 (62.7%)					
Body mass index (kg/m ²)	27.7 (4.9)					
Waist circumference (cm)	95.9 (14.4)					
Hypertension	294 (63.0%)					
Diabetes	64 (13.7%)					

Data descriptor: [Dryad Data -- Uncovering genetic mechanisms of hypertension through multi-omic analysis of the kidney](#)

Gene expression and alternative splicing data are derived from poly-A RNA-sequencing.

DNA methylation data are derived from the Illumina Infinium HumanMethylation450 BeadChip.

All data sets are the normalised values used for QTL testing with FastQTL.

All values have been quantile normalised and transformed by the rank-based inverse normal method.

Gene expression

Gene IDs map to ensembl v83

Values are normalised and transformed $\log_2(\text{TPM}+1)$ expression at the gene-level, calculated by Kallisto.

Alternative splicing

Intron excision isoform IDs map to GRCh38 coordinates.

Values are normalised and transformed intron usage ratios calculated by Leafcutter.

DNA Methylation

CpG IDs map to the Illumina IDs supplied in the Infinium HumanMethylation450 BeadChip manifest file.

Values are normalised and transformed M-values calculated by the R package minfi